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OCCUPATIONAL MELANOSIS FROM PITCH

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In a paper presented at the meeting of the American Dermatological Association in 1938 we [1] reported on our observations and investigations concerning the photosensitizing properties of pitch and its components. The present paper is a report on melanosis and dermatitis occurring in industries whose workers are exposed to pitch and tar. Histologic studies are recorded. Because of the paucity of reports on this subject in English and American publications, a review of pertinent literature is included.

This investigation was initiated at factory A in Wisconsin because there a troublesome and persistent dermatitis and melanosis had become an economic problem to the employer and were upsetting the morale of the employees. A similar factory in New York and others engaged in related industries in Pennsylvania were subsequently investigated by one of us (L. S.). More than 500 men were examined, and more than half of these had melanosis or premelanotic dermatitis. There were also many who had miscellaneous cutaneous lesions, such as pitch comedos, keratoses, folliculitis and furunculosis.

At factory A it was observed that the condition was confined to handlers of pitch or pitch products. Most of the employees were fair skinned and of German descent; some were Irish. They lived on farms or in neighboring villages. While some men were affected more severely than others, apparently none sufficiently exposed was immune. The most severe dermatitis and most intense pigmentation were observed in men employed outdoors at cutting, polishing and stacking finished conduits. When an indoor worker was placed in the yard dermatitis invariably developed; when he returned to indoor work it subsided,

* From the United States Public Health Service.

1. Foerster, H. R., and Schwartz, L.: Industrial Dermatitis and Melanosis Due to Photosensitization, Arch. Dermat. & Syph. **39**:55 (Jan.) 1939.

though the pigmentation persisted. Although the indoor work necessitated direct contact with pitch or pitch vapors and exposure to high temperatures, little or no dermatitis was manifest. On change of occupation the dermatitis usually subsided in approximately two weeks, and the pigmentation faded out in three or four months.

Factory A employed 65 men. Twenty-four cases of dermatitis, involving most of the yard workers, were recorded by the manager in 1936 and 1937, and these occurred throughout the year, exhibiting a greater prevalence from October to April than from May to September. The dermatitis involved chiefly the face and neck and was frequently associated with conjunctivitis, both conditions being most troublesome on bright sunny days, especially when there was snow on the ground or a strong wind. Tolerance, with persistent pigmentation, developed in some of the men, but most of them had to be rotated at various jobs, and certain new employees reacted so severely that they were considered unsuited for the work. In addition to dermatitis, hyperpigmentation and conjunctivitis, frequent complaints were made of pitch comedos on the face, hands and forearms, folliculitis and occasional furunculosis. Twenty of the men working in the yard and impregnating rooms were examined, and nearly all showed pitch comedos and acneform folliculitis of the face, forearms and hands, other cutaneous areas being clear. All of them showed considerable diffuse pigmentation of those surfaces exposed to sunlight, considerably darker than an ordinary sun tan, and no hyperpigmentation in covered areas. Some of the yard men showed erythema and desquamation of the face, particularly about the nasal alae, and some had labial herpes and cheilitis. A few workers had inflamed and swollen faces, with particular involvement of the eyelids and lips, and several had on the hands and forearms keratoses and papillomas of the type often seen in tar and petroleum workers. A few miscellaneous nonoccupational cutaneous diseases were noted, but no epitheliomas were observed in this factory.

Another conduit factory, B, in New York, was subsequently investigated. It was found that most of the employees had definite hyperpigmentation of the exposed surfaces of skin and that they were particularly susceptible to sunburn. There were many cases of pitch folliculitis, acne and furunculosis and several cases of epithelioma.

At factory C, in Pennsylvania, where the men were engaged in making roofing materials and in extracting coal tar oils, dermatitis was observed among handlers of both coal tar and coal tar pitch. Pitch dust was observed to be the most irritating, especially as used in the making of fuel briquets, and pitch dust and sunlight were definitely provocative of inflammation of the skin. Several cases of tar acne, tar keratoses and dermatitis were observed, and most of the workers showed a distinctive facial pigmentation.

A fourth investigation was carried out at plant D, where the pitch used by the conduit factories was made. It was learned that workers engaged in handling fuel pitch and exposed to pitch dust but not to vapors, those exposed to both vapors and dust in melting roofing pitch, those engaged in extracting pitch oils by the further heating of fuel pitch and those exposed to the fumes of hot road tar while making roads were all subject to dermatitis and pigmentation and were particularly susceptible to sunburn. Some of the workers were so highly photosensitive that they could not be employed in the day time, but worked satisfactorily on night shifts. It was observed that men on night shifts did not suffer much from sunburn if they cleansed their skins thoroughly with soap and water before going off duty and that these men could be exposed to sunlight before returning to work without dermatitis developing on subsequent contact with tar. In a subsidiary plant where coke was made from pitch and all operations were conducted indoors the workers were not affected.

REPORT OF CASES

The histories of 2 cases of typical dermatitis at factory A, the subject of most of the investigative work, will be stated briefly as examples:

CASE 1.—H. D., a white man aged 26, was first employed in March 1937 and assigned to yard work. He had not had any cutaneous disease previously and was in normal health. On his first day at work, cutting and stacking conduits, after ten hours in the sunlight and cold wind his face burned and was diffusely red, as after sunburn. His face remained red for three days and then peeled. He continued at his work, and the sunburn-like inflammation and subsequent peeling of the skin recurred at variable intervals, usually twice weekly, and in such intensity that he could not shave while the inflammation was at its height. Tanning of the face was not observed by him until two months later. The dermatitis was usually most intense about the nose, mouth and malar areas and was frequently attended by conjunctivitis. The hands and forearms were moderately involved, and the unprotected part of the neck was inflamed. Some protection and relief were obtained by the wearing of a cap and goggles and the application of various salves. Most irritation and tenderness were noticed in hot weather during profuse sweating, on windy days and when the ground was covered with snow.

The face and neck with the exception of the forehead were diffusely and uniformly reddish brown, except for the markings of blood vessels, and this process was most intense on the sides of the neck, on the malar areas and about the mouth and nose. The backs of the hands and forearms were diffusely brown as with sun tan. The pigmentation was not mottled or reticulated and not concentrated about the follicular openings. Numerous pitch comedos were present on the forearms and hands. There were no keratoses or folliculitis. The skin of the covered parts of the body, including the axillas and groins, was pale and normally pigmented. The mucous membranes were normal except for congestion of the conjunctivas. The scleras were not discolored.

CASE 2.—E. B., a white man aged 33, brunet, had been employed for eight years and had had dermatitis recurrently during all this time. He was the

plant forearm and usually spent half his time outdoors. He had occasionally been employed indoors continuously for several weeks, and at such times there had been no dermatitis. After adequate exposure to pitch dust and sunlight he first observed diffuse redness of the face, associated with sensations of burning and smarting, and as the process increased, swelling occurred. Desquamation was observed on about the third day, and this sequence of events occurred about twice weekly, even though the hyperpigmentation persisted. The neck had been only slightly involved, and the hands had never been affected.

The most striking feature on examination was a diffuse and intense grayish brown discoloration of the face, especially about the mouth, cheeks and chin, and of the upper part of the neck. Dermatitis and moderate desquamation were observed in the malar areas, about the mouth and on the chin. The scleras were discolored as in mild jaundice, and the conjunctivas were injected. The scalp was unaffected. The buccal mucosa and the covered parts of the body were of normal color and showed no areas of hyperpigmentation. Cutaneous atrophy, keratoses, papillomas and folliculitis were absent, and there were no features of "sailor's" or "farmer's" skin other than the hyperpigmentation, and this had an unnatural grayish cast. This is significant in view of the eight years of recurrent dermatitis and the reports by Hoffmann, Habermann and others of cutaneous atrophy and hyperkeratosis with tar or pitch melanosis.

REVIEW OF THE LITERATURE ON DERMATITIS AND MELANOSIS DUE TO TAR AND PITCH

The first observations of occupational dermatitis due to pitch and tar are credited to Volkmann, who in 1875 accurately described the various cutaneous changes due to these substances. Ehrmann [2] subsequently elaborated on these observations and described three types of cutaneous reaction to pitch: (1) dermatitis with hyperkeratoses and papillomas, (2) pitch comedos and folliculitis and (3) melanosis. He aptly designated the last *Indianerhaut* but failed to recognize photosensitization as its cause. He concluded that the melanosis was the consequence of intense hyperemia and irritation of the skin that also resulted in infiltration. He expressed the belief that the scleras were discolored by pitch vapors but that the skin was not discolored in a similar manner. He observed that this condition was confined to the face and neck and the exposed parts of the arms.

Lewin [3] appears to have been the first author to associate pitch with a possible photodynamic activity. In this he based his deductions on the earlier experimental work of Raabs [4] on the effect of fluorescent substances on Infusoria. He observed 100 cases among the employees of a cable works engaged in impregnating paper tubes with hard coal tar pitch; the condition was confined to exposed parts and varied from erythema to dermatitis, associated with sensations of burn-

2. Ehrmann, O.: Monatsh. f. prakt. Dermat. **48**:18, 1909.
3. Lewin, L.: München. med. Wchnschr. **60**:1529, 1913.
4. Raabs, O.: Ztschr. f. Biol. **39**:537, 1900.

ing and itching that were most severe when the patient ventured into the sunlight and subsided at night. It is noteworthy that he did not mention the occurrence of pigmentation. Lewin suspected a fluorescent ingredient of the coal tar residue had a photodynamic action on the skin, thereby causing dermatitis, and he pointed out that acridine was such a substance. He did no experimental work, and he concluded with the remarks that this dermatitis might be a direct contact dermatitis or might be the effect of absorption, either directly through the skin or by inhalation of vapors, of a photodynamic substance.

Herxheimer and Nathan [5] reported dermatitis solare following photosensitization from "carboneol," a solution of coal tar in carbon tetrachloride and chloroform. This condition was an acute vesicular and papular dermatitis that occurred in sharply demarcated elevated edematous plaques on light-exposed areas only. Melanosis apparently did not occur. The ultraviolet rays of sunlight were suspected of being the sensitizing radiation, but no experiments were made to confirm this opinion.

In 1917 Riehl [6] described a peculiar pigmentary disorder of the skin, and his observation resulted in subsequent confusion of the condition with tar melanoses. Riehl's patients were men, women and children of indoor habits and activities, observed in Vienna in the winter and spring, and exhibiting a diffuse dark brown—at times bronze or grayish brown—discoloration involving chiefly the forehead and sides of the face. The affected skin was thickened, and the neck and in some cases the hands, extensor surfaces of the forearms, axillary folds, mammae and umbilical and inguinal areas were likewise involved. The pigmentation appeared gradually, without evidence of preceding inflammation. While the appearance in most cases suggested the tanning of sunlight, there had been little exposure to sun, and the pigmentation was not sharply limited by the clothing and was definitely and slowly progressive. There was no record of contact with tar preparations. Riehl concluded that this disease was caused by a toxemia of nutritional origin, probably resulting from the use of horse beans added to flour and the substitution of mineral grease for butter, possibly sensitizing the affected persons to the pigmentary reactions of sunlight.

The following year (1918) Hoffmann and Habermann [7] described in patients who had been in direct contact with tars, oils and greases a melanodermatitis which they attributed to the action of medicinal and occupational tar substitutes for the petroleum products not obtainable on account of the war blockade.

5. Herzheimer, K., and Nathan, E.: Dermat. Ztschr. **24**:385, 1917.

6. Riehl, G.: Wien. klin. Wchnschr. **30**:780, 1917.

7. Hoffmann, E., and Habermann, R.: Deutsche med. Wchnschr. **44**:261, 1918.

In Hoffmann and Habermann's cases there was a macular or mottled, at times somewhat reticulated, pigmentation in various shades of brown to slate gray. This melanosis was particularly well defined on the arms, where it was sometimes configurated or lichenoid, and it was not uniformly diffuse, like Riehl's, or sharply demarcated by the clothing, as in Ehrmann's cases. Small petechiae were noted as early manifestations, and some patients showed follicular keratoses, comedos and warty proliferations, but the pigmentary changes predominated. The authors described a hyperkeratotic follicular pigmented type, attributed to contact with tar and pitch contaminants of lubricating oils, paraffins, greases and petrolatum preparations, and a toxic melanodermatitis, ascribed to inhalation of vapors containing tar or pitch. They suspected that heat or light rays play a part in the pigmentation, in either its causation or its intensification and subsequently recognized light sensitization as a causative or contributing factor, with acridine as the possible photosensitizing agent. No investigation was made of this subject.

Meirowsky [8] reported a case of melanosis of the face resulting from contact with "goudron," a tar distillate and paraffin preparation used for insulating walls against moisture. He attributed the melanosis to the inhalation of tar vapors by persons photosensitized by the ingestion of food substitutes.

Blaschko [9] called attention to the marked increase in these melanoses after the onset of the war, particularly in industry, and they became known as the "war melanoses." A distinction was made between those described by Riehl and those by Hoffmann and Habermann.

Habermann [10] wrote exhaustively on this subject, considered both endogenous and exogenous sources of sensitization to light, chiefly by acridine, and stated that in all cases the melanosis was fundamentally an inflammatory toxic process. Habermann stated that this condition and Riehl's could not be sharply differentiated histologically, and on the basis of histologic as well as clinical study he favored Hoffmann's designation, melanodermatitis toxica lichenoides, for all cases.

Koelsch [11] and Ullmann [12] have both written extensively on the occurrence of dermatoses in the tar and naphtha distilling industries. In spite of fundamental chemical differences in the two types of distillates, they were observed to cause similar dermatoses, which were grouped in three fundamental clinical types: (1) follicular lesions

8. Meirowsky: Arch. f. Dermat. u. Syph. **25**:378, 1918.
9. Blaschko, A.: Dermat. Ztschr. (supp.) **26**:2, 1918.
10. Habermann, R.: Dermat. Ztschr. **30**:62, 1920.
11. Koelsch, F.: Zentralbl. f. Gewerbehyg. **7**:157, 1919; in Ullmann,[12] p. 303.
12. Ullmann, K.: Die Schädigungen der Haut durch Beruf und gewerbliche Arbeit, Leipzig, Leopold Voss, 1926, vol. 2, p. 226.

produced by both mechanical plugging and inflammation, (2) epidermal proliferations, such as keratoses, verrucae, papillomas and epitheliomas, and (3) melanoses. The melanoses were found particularly after inflammation, apparently dermatitis venenata, produced by lubricating oils.

Referring to the melanoses, Koelsch expressed disagreement with those authors who attributed the pigmentation to the inhalation or swallowing of specific toxic agents and with those who blamed nutritional disturbances or internal toxemias of a pellagroid nature. Koelsch stated that when the skin is sensitized by derivatives of coal tar it is susceptible to the action of light and becomes inflamed, pigmentation resulting on subsidence of the dermatitis. The American pitches originating from petroleum, used before the war, were thought to be less harmful in this regard.

Koelsch described the intermittent character of the inflammatory reactions of pitch and asphalt workers and observed that the symptoms of erythema, edema and pruritus, confined to exposed parts, were of only moderate severity in diffuse light and became intensified, at times unbearably so, in direct sunlight or reflected light from snow. Koelsch observed that many of the workers with melanoderma had no subjective symptoms and had observed only yellowish to brownish discoloration of the skin, without noticeable preceding inflammation. The pigmentation was usually diffuse but occasionally retiform or macular. The forehead, face and neck were always involved, the covered parts occasionally. The scleras were frequently yellow, and keratoses were sometimes present.

Koelsch also reported on pigmentation of the face and keratoses and folliculitis on exposed areas and parts in contact with contaminated clothing, occurring in workers with carbolineum (crude anthracene oil).

Arnstein [13] recorded tar melanoses in female employees of a dry battery factory where tar was used as a packing material and where the workers were exposed to tar fumes as well as to direct contact with tar in a poorly ventilated room. He suggested that nutritional disturbances might predispose to pigmentation, though he expressed the belief that impurities in the tar and paraffin products, as suggested by Koelsch, might have been factors, but he did not comment on exposure to light as a possible etiologic factor. In addition, many other cases of melanosis from exposure to coal and tar and their derivatives have been reported by German authors. A few have been reported in the French and British literature.

13. Arnstein, A.: München. med. Wchnschr. **67**:902, 1920.

O'Donovan [14] observed melanosis in outdoor workers handling crude anthracene while men engaged indoors in packing the distillates were unaffected.

Kistiakowski [15] reported photosensitization dermatitis and pigmentation of exposed parts, comedos and folliculitis in the making of artificial asphalt from coal tar, anthracene oil and coal tar pitch. He ascribed the photosensitization to the inhalation of vapors and the swallowing of dust containing anthracene, acridine and phenanthrene.

Wieder [16] reported an occupational melanosis of the face in a chemist who, on experimental testing, reacted photochemically to both ordinary naphthalene and benzanthrone-anthraquinone when exposed to artificial ultraviolet rays, but who exhibited no reactions when these rays were excluded. Tests were negative as regards pigmentation with chemically pure naphthalene and a number of other chemicals with which the patient was repeatedly in contact. The history indicated a definite association of mild dermatitis and subsequent melanosis, particularly with exposure to the fumes of naphthalene in sulfonation. Wieder cited Ullmann as stating that while Bayet and others attributed pigmentation from tar and pitch to the arsenic contained in coal, Vossenaar demonstrated that coal workers in whom "pitch skin" was uncommon showed four times as much arsenic in the urine as workers in a briquet factory, in whom pitch skin is common. Other investigators have supported Vossenaar, among them Filigu, who produced tar and pitch skin with arsenic-free tar.

Kinnear [17] reported in a jute spinner melanosis which he described as a dark brown macular pigmentation of the face with an associated oil folliculitis of the arms, forearms and legs. He described the condition as being similar to that reported by Hoffmann and Habermann, though he called it Riehl's melanosis. The pigmentation in Kinnear's case was reticular in distribution, with pale areas corresponding to follicular openings. Hoffmann and Habermann, Riehl and Kinnear reported the observation of atrophy, as well as of hyperkeratosis and reticulation, and though atrophy was never marked, it induced Kinnear to group the condition with the poikilodermas. He likewise included Civatte's *poikilodermie réticulée pigmentaire du visage et du cou* and differentiated this group from Jacobi's type of poikiloderma, which exhibits more extensive atrophy, less pigmentation and a characteristic telangiectasia.

14. O'Donovan, W. J.: Cancer of the Skin Due to Occupation: Tar Carcinoma, Arch. Dermat. & Syph. **19**:595 (April) 1929.

15. Kistiakowski, E. W.: Ann. de dermat. et syph. **1**:63, 1930.

16. Wieder, L. M.: Occupational Melanosis, with Report of a Case, Arch. Dermat. & Syph. **25**:624 (April) 1932.

17. Kinnear, J.: Brit. J. Dermat. **47**:191, 1935.

None of Civatte's patients were known to have been in contact with oil or tar, but Civatte expressed the suspicion that the condition he reported was in some way the result of photosensitization.

HISTOPATHOLOGY

Three specimens for histologic study were removed from H. D. One was taken from an area of pigmentation on an exposed portion of the neck that had been tested with pitch and exposed to daylight ten days previously and had reacted with a sharp erythema and edema; the second, from an adjacent exposed and tanned area, of previous pitch melanosis that had not been subjected to a patch test, and the third, from a small area of pigmentation on the abdomen that had developed after a mild erythema following repetition of a patch test with pitch and ultraviolet rays, without the production of erythema or pigmentation in the surrounding normal skin.[19]

The sections from these three specimens showed similar changes in varying degree. There were hyperkeratosis, an increase in the granular layer, intercellular edema in the rete, edema and dilated vascular spaces in the papillae, subpapillary layer and midcutis and a moderate perivascular infiltrate about the vascular structures, chiefly of endothelioid and round cells. The silver stain showed an abundance of fine pigment granules in and among the cells of the basal layer and numerous small aggregations of pigment-containing cells in the upper and the middle portion of the cutis, chiefly in the vicinity of the vessels and follicular structures but widely distributed.

The first biopsy specimen showed more edema and inflammatory change than the others, and the third, a greater amount of pigment in the corium. The amount of pigment in all sections studied was moderate, and this may be accounted for by the short intervals between the patch tests and the removal of the specimens and by the absence of sunshine at the time the tests were made.

Meirowsky[20] studied 8 cases of tar melanosis histologically. In most of them there was little or no pigment in the epidermis, but in all there was an abundance of pigment in the corium, demonstrable by silver stain but giving a negative reaction to the dopa test. In the case of a pitch worker with melanosis of the face and body the cells of the basal layer showed sparse but definite pigmentation, with underlying slight pigmentation in the uppermost layer of the corium. Beneath this area was a dense layer of heavily pigmented connective tissue cells, so densely packed with coarse pigment granules that only their walls could be

18. Footnote deleted on proof.

19. Dr. S. W. Becker prepared the specimens.

20. Meirowsky: Virchows Arch. f. path. Anat. **255**:303, 1925.

defined. With few exceptions, they did not exhibit dendrites. The pigmented cells were grouped about the lymph spaces or distributed in clusters and singly throughout the corium. Some groups of pigment cells were so dense as to simulate a melanotic neoplasm. These histologic observations were similar to those of Lipschutz [21] in mice that had been painted with tar.

In cases of Riehl's [6] melanosis there were no inflammatory changes in the epidermis and little pigment, even in the corium. The latter showed a sharply defined round cell infiltrate directly below the papillae and intensely pigmented chromatophores in a horizontal band below the papillae, in clusters and scattered about the corium. The infiltrate extended deeper only about the follicles, and moderate edema and little infiltrate were noted in the papillae.

In Hoffmann and Habermann's [7] cases of toxic melanodermatitis the cells of the basal layer and the lower part of the rete showed little or no pigment, and the upper part of the rete and the stratum corneum were free of pigment, while in the papillae and cutis proper there was an increase of chromatophores, arranged in groups or sparsely distributed directly beneath the epidermis. There were hyperkeratosis, no parakeratosis, a well developed stratum granulosum and in some cases constriction of the rete and epidermal dystrophy. There were infiltrates about the follicles, but the middle and the deep part of the cutis were otherwise unchanged.

In hyperkeratosis follicularis pigmentosa these authors observed pronounced pigmentation of the epidermis and an increase of large chromatophores, associated with edema and a mild inflammatory perivascular reaction in the papillary and subpapillary layers of the corium. There were also large follicular horny plugs.

Kissmeyer [22] noted edema of the epidermis and papillae, little pigment in the epidermis and a marked accumulation of pigment cells in the upper part of the cutis, with a moderate inflammatory infiltrate, both perivascular and diffuse, and a positive dopa reaction in the basal layer.

In Wieder's case of occupational melanosis the epidermis showed moderate follicular hyperkeratosis and slight intracellular edema, with considerable pigmentary activity in the melanoblasts of the basal layer and an abundance of pigment in the chromatophores of the upper part of the cutis. A moderate amount of pigmentation was observed in the rete. There were no marked changes in the vessels or the connective tissue indicative of inflammation, which conformed to the clinical findings.

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21. Lipschutz, cited by Meirowsky.[20]

22. Kissmeyer, A.: Arch. f. Dermat. u. Syph. **140**:357, 1922.

MELANIN AND MELANOSIS

Normal pigmentation and hyperpigmentation of the skin are due to the presence of an organic dye, a pyrocatechin derivative, melanin, which is found chiefly in the spider-like dendritic cells interposed between the cuboidal cells of the basal layer, but may also be present elsewhere in the epidermis and in the corium. The origin of the dendritic cells is not definitely known. Peck [23] supported the view of Bloch that they represent special functional phases of ectodermal pigment-producing basal cells, while Becker [24] expressed the belief that they are normally present in the basal layer and assume the pigmented dendritic form under the influence of pigment production, and Masson claimed to have demonstrated their genesis from nerve tissue of the Langerhans cell type.

Pigment production is a complex process. It was exhaustively studied by Bloch.[25] The epidermal dendritic cells of the basal layer and hair follicles are called melanoblasts and are believed to produce melanin from a colorless propigment, melanogen. The latter is probably derived from a polycyclic amino acid, such as tyrosine, by action of an enzyme, such as tyrosinase, and is taken up by the melanoblasts. These cells then produce the colored melanin granules within their protoplasm either by simple oxidation of the melanogen or by the interaction of a specific catalytic ferment or enzyme, such as oxidase, thought to be normally present. This ferment is apparently activated by a specific radiation of light. The colorless melanogen may be present in the cell or may be taken up by it at the time of pigment production. Bloch called it an epinephrine-like chemical, 3,4-dioxyphenylalanine, or dopa, and he designated the activating ferment dopa oxidase and demonstrated the specificity of the reaction.

Kissmeyer [22] called attention to the large number of polycyclic hydrocarbons known to be present in coal tar and theorized that it may contain one or more as yet unidentified substances chemically closely related to dioxyphenylalanine that may act as propigment activators and react with a pigment oxidase in the production of melanin or a melanin-like substance. He cited a somewhat analogous purely physical reaction described by Kreibich in which paradimethylphenylenediamine served as the reagen.

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23. Peck, S. M.: Pigment (Melanin) Studies of Human Skin After Application of Thorium X, with Special Reference to the Origin and Function of Dendritic Cells, Arch. Dermat. & Syph. **21**:916 (June) 1930.

24. Becker, S. W.: Melanin Pigmentation: A Systematic Study of the Pigment of Human Skin and Upper Mucous Membranes, with Special Consideration of the Pigmented Dendritic Cells, Arch. Dermat. & Syph. **16**:259 (Sept.) 1927.

25. Bloch, B., in Jadassohn, J.: Handbuch der Haut- und Geschlechtskrankheiten, Berlin, Julius Springer, 1927, vol. 1, pt. 1, p. 434.

Mayer [26] expressed the belief that there is an intermediate step in the oxidation of melanogen into melanin, the first being the production of a quinone, and that it combines with a protein, albuminoid or lipoid, the color of the pigment varying with the combination. Mayer stated that he did not consider dopa essential for the production of melanin.

While it appears that the biochemical reaction of melanin production is an oxidation process, Meirowsky expressed the belief that he had demonstrated pigmentation without the action of an oxidizing ferment, apparently through the effect of heat or of ultraviolet rays.

Melanin may be produced in the absence of light, Becker [24] having demonstrated its presence in dendritic cells in the buccal and pharyngeal mucosa. It is found also in mongolian spots, in internal melanotic tumors and on covered parts in postinflammatory pigmentation.

In the cutis melanin is found in spindle-shaped connective tissue cells called melanophores or chromatophores. These are phagocytic cells that carry the pigment from the dendritic cells to the blood vessels. Meirowsky [8] expressed the opinion that under certain conditions, such as prevail in tar melanoses, the melanophores may become self producers of melanin. In his cases of tar melanosis the melanin deposits were pronounced in the corium, while scanty or absent in the epidermis. In experimental tar painting of mice, Lipschutz [21] observed pigmentation in the corium forty hours after a single application, and he concluded that the epidermal cells could not produce melanin in that time and transmit it to the corium and that therefore it was produced by the cells of the corium.

PREVENTION AND TREATMENT

The most important and effective procedure in combating pitch dermatoses is a problem in sanitary engineering. It was observed by us that medicinal applications, both therapeutic and protective, were unsatisfactory in ameliorating this dermatitis and ineffective in eliminating it. The installation of ventilators, the discontinuance of operations creating excessive pitch dust, particularly outdoors, and other engineering improvements, such as enclosing certain procedures, all carried out at the instigation of one of us (L. S.), reduced to a minor hazard what had been a major one threatening the successful operation of the factories. Likewise of importance is the matter of personal hygiene of the workers, which can be effectively enforced by providing adequate locker space and requiring daily shower baths and daily change to clean work clothes. Supervision by an industrial nurse with authority to enforce rules of sanitation and hygiene is essential.

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26. Mayer, R. L.: Klin. Wchnschr. **7**:2471, 1928.

In our first paper we reported that our investigations indicated that dermatitis and melanosis from pitch are due to its photosensitizing properties, and that the inherent photosensitizers are the aromatic hydrocarbons, inclusive of acridine and anthracene, and possibly also as yet unrecognized photosensitizers, such as derivatives of chlorophyll. The use of chemical blockers of light in applications to exposed cutaneous surfaces is therefore of importance in both therapy and prophylaxis.

As protective creams we have found preparations of resorcinol and quinine satisfactory; of these the following formula is an example:

	Parts
Quinine hydrochloride	0.02
Stearic acid	2.9
Wool fat	8
Liquid petrolatum	5
Triethanolamine	0.38
Ethyldiethyleneglycol (carbitol)	1
Terpineol	0.01
Distilled water	10

In the treatment of pitch dermatitis we provided relief with protective and sedative lotions containing ichthammol and zinc oxide for application during working hours and soothing emulsions or creams for application at night.

For protection against acridine sensitization Hausmann and Haxthausen [27] cited Janison, who advised oral administration of 0.25 Gm. of resorcinol daily. Janison stated that resorcinol acts as an antiphotocatalyst but that it may cause gastric disturbances and dermatitis. He stated that pyrocatechin (orthodioxybenzene) is superior to resorcinol because its greater solubility allows it to be used intravenously. The observations that pigmented animals are not photosensitive and that in fagopyrism only the vitiliginous areas show inflammation suggest that therapeutic exposures to ultraviolet rays to build up a defensive melanosis of the face and neck may have some practical application in persons who are unable to avoid the acquisition of dermatitis.

CONCLUSIONS

1. Dermatitis and melanosis are important occupational hazards in coal tar and pitch industries.

2. Most workmen exposed to this hazard have either dermatitis or melanosis, though some are not affected, and variations in susceptibility exist.

27. Hausmann, W., and Haxthausen, H.: Die Lichterkrankungen der Haut, Sonderbände zu Strahlentherapie, Berlin, Urban & Schwarzenberg, 1929, vol. 11, p. 1.

3. Histologic studies indicate that the discoloration due to pitch is true melanosis.

4. We have previously reported that pitch dermatitis and melanosis are true reactions of photosensitization resulting from the local exogenous activity of a specific photosensitizer in pitch, activated by specific spectral bands of light.

5. We believe that pitch dermatitis can be eliminated as a hazard to indoor workers by effective sanitary engineering and by provision for and enforcement of thorough personal hygiene.

6. By similar means we believe that it can be materially reduced and its severity ameliorated among outdoor workers and that where exposure to pitch and tar dust and vapors cannot be avoided the incidence of dermatitis can be reduced by night operations, rotation of work and the selection of a tolerant personnel.

7. Appropriate protective and therapeutic measures include the application of creams and lotions containing physical and chemical blockers of light.

ACETARSONE THERAPY IN ONE HUNDRED AND EIGHTY-SEVEN CASES OF CONGENITAL SYPHILIS

WITH OBSERVATIONS ON A GROUP OF EIGHTY-SEVEN PATIENTS RECEIVING NO TREATMENT

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Since 1931, administration of acetarsone by mouth has been the principal method of treatment in cases of congenital syphilis observed in the Sigma Clinic of the Children's Hospital. At the time of this report 196 patients have received this drug, and for 187 of these we believe that the diagnosis of syphilis was correct. We have followed the 187 cases for periods varying from two months to six and one-half years. The present study is a review of these cases, particularly in regard to the incidence of reactions to treatment, the effect of treatment on the Wassermann and the Kahn reaction of the blood, the incidence of clinical and serologic relapse in cases followed for a year or more and the incidence of neurosyphilis. The average period during which these cases were followed after treatment was begun was two and eight-tenths years. The average period of observation of the same cases since the admission to the outpatient department was four and four-tenths years.

In 87 cases in this series the diagnosis of syphilis was not made in the dispensary at the initial examination and some subsequent examinations, and in this group the average period between registration of the patient and recognition of the presence of syphilis was three years. This has afforded us an opportunity for analysis of a considerable number of cases in an effort to determine the reasons for failure to make an initial diagnosis of syphilis and the incidence of active lesions of syphilis after the institution of medical supervision. It is our feeling that a consideration of the reasons for failure to recognize syphilis in general pediatric practice is of far greater importance than a discussion of par-

A portion of the acetarsone used in this study was furnished by Merck & Co., Rahway, N. J.

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ticular methods of treatment, since the cure in the majority of cases is absolutely dependent on early diagnosis.

Acetarsone was originally synthesized by Ehrlich and Hata,[1] but apparently was discarded by them as too toxic. In 1921, Fourneau[2] revived the drug as an antisyphilitic agent and stimulated trials of it in treatment of various aspects of the disease. Since then over forty reports of the effect of acetarsone on congenital syphilis have appeared. We have reviewed most of these papers, but they have been amply summarized by several writers,[3] and we shall confine our reference to them to certain aspects of our report. Whipple and Dunham[4] stated that in only four studies has the period of observation been sufficient for any adequate evaluation of acetarsone. The most extensive series in which the cases have been well followed, consisting of 65 cases observed for three years, has been reported by Traisman.[3d]

The status of acetarsone as a preventer of late congenital syphilis has by no means been settled. Recently its use for man has been severely criticized by Rosahn and Kemp,[5] editorially by the *American Journal of Syphilis, Gonorrhea and Venereal Diseases*[6] and by Cole.[7] On the other hand, American pediatricians have uniformly reported favorably on the drug, and Traisman[8] and Rosenbaum[9] have answered

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1. Ehrlich, P., and Hata, S.: Die experimentelle Chemotherapie der Spirillose, Berlin, Julius Springer, 1910.

2. Fourneau, E.: Sur l'emploi de l'acide oxyaminophénylarsinique et des acides arylarsiniques en général dans le traitement des spirilloses et des trypanosomiases, Ann. Inst. Pasteur **35**:571-574, 1921.

3. (*a*) Muller, E.: Die Spirozidbehandlung der angeborene Syphilis und ihre bisherigen Erfolge, Arch. f. Kinderh. **91**:108-124, 1930. (*b*) Nedelmann, E.: Erfahrungen mit der Spirozidbehandlung der Lues congenita, Jahrb. f. Kinderh. **134**:89-101, 1932. (*c*) Gerth, R.: Zur Frage der Heilung der kongenitalen Lues des Säuglings mit kleinen Spirociddosen, Monatschr. f. Kinderh. **67**:46-58, 1936. (*d*) Traisman, A. S.: Further Observations on the Use of Acetarsone in the Treatment of Congenital Syphilis, J. Pediat. **7**:495-511, 1935.

4. Whipple, D. V., and Dunham, E. C.: Congenital Syphilis: II. Prevention and Treatment, J. Pediat. **13**:101-119, 1938. This study is the most complete, current review of congenital syphilis available.

5. Rosahn, P. D., and Kemp, J. E.: The Oral Administration of Stovarsol in the Treatment of Experimental Syphilis of the Rabbit, Am. J. Syph., Gonor. & Ven. Dis. **21**:180-198, 1937.

6. The Oral Medication of Syphilis, editorial, Am. J. Syph., Gonor. & Ven. Dis. **21**:456-461, 1937.

7. Cole, H. N.: The Pharmacopeia and the Physician: The Use of Antisyphilitic Remedies, J. A. M. A. **107**:2123-2131 (Dec. 26) 1936; The Use of Antisyphilitic Remedies, ibid. **108**:825, (March 6) 1937.

8. Traisman, A. S.: The Use of Antisyphilitic Remedies, J. A. M. A. **108**: 825 (March 6) 1937.

9. Rosenbaum, H. A.: Acetarsone in the Treatment of Syphilis, J. A. M. A. **108**:1280 (April 10) 1937.

Cole's criticisms vigorously. Clinical experience with acetarsone has in this country been largely acquired by pediatricians.

The chief objections advanced to acetarsone therapy are that (1) the difference between the toxic and the therapeutic dose is so small and the variations in toxicity of various lots of the drug so great as to make the clinical use of acetarsone hazardous and unjustifiable; (2) experience with the drug has not yet progressed to a point at which any conclusions concerning its final antisyphilitic effect can be made, and the drug is therefore still experimental and should not be advocated for general use; (3) the results obtained with standard means of treatment are so good, particularly for infants, as to make another medicament superfluous, and (4) in many cases the physician cannot be certain that the drug is actually administered.

Among the arguments in favor of acetarsone therapy are that (1) the drug is easily administered without the psychic and physical trauma incident to parenteral injections; (2) the effect on the active lesions of syphilis is good; (3) the serologic response is as good as that obtained with "standard" preparations, and (4) the reactions are not ordinarily serious and may be reduced by avoiding excessive doses and discontinuing the drug at the first sign of intolerance.

We shall review briefly the chief evidence tending to support or disprove these arguments.

Rosahn and Kemp [5] have well summarized the conflicting evidence concerning the toxicity of acetarsone for animals. In an effort to obtain further data on this subject with the standard laboratory animal of experimental syphilis, the rabbit, these investigators administered varying doses of commercial acetarsone. They found that with a dose well within the range used for human beings, 20 mg. per kilogram of body weight daily, 4 of 6 rabbits died from the effects of the drug. When the dose was reduced to 15 mg. per kilogram one group of animals survived, while another group, treated later with the identical drug, showed a mortality of over 50 per cent as a result of acetarsone alone.

Raiziss [10] stated that he has noted a similar variation in the toxicity of acetarsone for rabbits and that his experience at present indicates that the maximum dose tolerated is between 25 and 50 mg. per kilogram. In recent experiments performed by him, however, he has found that differences in tolerance of acetarsone apparently exists in different species; that dogs may be given 100 mg. per kilogram of acetarsone by mouth for twelve treatments over a period of six weeks and that rats "tolerate 3 to 4 Gm. of acetarsone by mouth."

It is difficult to come to any conclusions concerning the incidence of toxic reactions to acetarsone in man on the basis of published

10. Raiziss, G. W.: Personal communication to the authors.

reports. Stokes [11] has tabulated the wide variety of untoward effects, and Whipple and Dunham [4] stated that 7 deaths directly attributable to the drug have occurred in over 1,000 recorded cases in which it was used.

Concerning other objections to the use of acetarsone in the treatment of man, we do not believe that a sufficient number of cases have been observed for adequate periods to justify final conclusions. In a recent study by the Cooperative Clinical Group [12] it has been shown that a patient with late congenital syphilis is subject to the risk of interstitial keratitis until the age of 25. The report also showed that while the incidence of interstitial keratitis is greatly reduced (from 36 to 2 per cent) by a large amount of previous standard treatment with arsphenamine and heavy metal, there remains an irreducible minimum of cases in which this complication will develop when treatment is not begun until the patient is 2 years of age.

The objection that acetarsone therapy may fail to be carried out through lack of cooperation from the parent in the administration of the drug is a real one, to our minds, and we believe that it has not received adequate attention. No study of this factor has been made, and it has deterred some competent workers from using acetarsone.[13]

With the objection that standard agents of treatment are so good for early congenital syphilis as to admit of no indication for a trial of others we are not in agreement. While the treatment of congenital syphilis has been advanced enormously in efficiency during the past two decades, its long-drawn-out course, its sometimes difficult technical requirements and the dangers of reaction involved in all methods represent a status far from perfection. Even with otherwise healthy patients with early infantile syphilis the serologic results with standard preparations are not entirely satisfactory (Smith [14]). The residuum of cases of late syphilis with an unsatisfactory outcome is clearly indicated in the study carried out by Cole and his associates.[12]

Of the advantages of acetarsone therapy, the ease of administration has been emphasized in almost all reports. We agree that this may be accepted as a cogent argument provided the clinical results are comparable with those obtained with other preparations. If the effect of

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11. Stokes, J. H.: Modern Clinical Syphilology: Diagnosis, Treatment, Case Studies, ed. 2, Philadelphia, W. B. Saunders Company, 1934.

12. Cole, H. N.; Usilton, L. J.; Moore, J. E.; O'Leary, P. A.; Stokes, J. H.; Wile, U. J.; Parran, F., Jr., and Vonderlehr, R. A.: Late Prenatal Syphilis, with Special Reference to Interstitial Keratitis: Its Prevention and Treatment, Arch. Dermat. & Syph. **35**:563-579 (April) 1937.

13. Smith, F. R., Jr.: Congenital Syphilis: Results of Treatment in Children, J. A. M. A. **105**:409-412 (Aug. 10) 1935.

14. Smith, F. R., Jr.: Congenital Syphilis in Children: Results of Treatment in Five Hundred and Twenty-One Patients, Am. J. Syph. & Neurol. **19**: 532-546, 1935; Am. J. Syph., Gonor. & Ven. Dis. **20**:45-55, 1936.

acetarsone on the course of syphilis is inferior, however, such an argument is highly fallacious, and persistence in such treatment on such grounds can, in our opinion, be regarded only as an evidence of professional indolence.

There can be no question that acetarsone is an active antisyphilitic drug. We have seen no report of failure of mucocutaneous lesions to respond. The rapidity of healing varies somewhat and has been expressed in general terms in most reports. The average is between two and four weeks.[15] The evidence of the effect of acetarsone on acute syphilis of the bone, presented by Traisman [3d] and by Friedman [16] is convincing and indicates that healing is usually complete after one course (nine weeks with the Bratusch-Marrain system). In an earlier survey [17] we noted a prompt response in 11 cases of syphilis of the bone. Yampolsky,[18] however, has reported rather disappointing results in this regard.

MATERIAL STUDIED

In the present series of 196 patients given varying amounts of acetarsone we believe the diagnosis of syphilis to have been valid for 187. Of this number, 87 have been followed for more than three years since treatment was started, 116 over two years and 145 over one year. Eighty-three per cent of the patients were Negroes. The serologic follow-up has been good, adequate data as to the final status when the patient was last seen are available, and examinations of the spinal fluid have been done in 102 cases after some treatment. The observations on cutaneous lesions were not recorded frequently enough to permit of more than a general impression as to the rapidity with which they disappeared under treatment with acetarsone. The roentgen follow-up of cases of osseous lesions is incomplete. Observation of reactions to treatment was especially careful.

DOSAGE

The scheme of dosage suggested by Maxwell and Glaser [15b] was followed. For a few young infants the initial dose was reduced one half. In table 1 this system is compared with the widely used schedule suggested by Bratusch-Marrain,[19] in which generally smaller doses based

15. (*a*) Rosenbaum, H. A.: Stovarsol in the Treatment of Syphilis in Infants and in Children, Am. J. Dis. Child. **44**:25-30 (July) 1932. (*b*) Maxwell, C. H., and Glaser, J.: Treatment of Congenital Syphilis with Acetarsone Given by Mouth, ibid. **43**:1461-1489 (June) 1932. (*c*) Baumbach: Ueber Erfahrungen mit der Spirocidkur bei Lues congenita, Arch. f. Kinderh. **99**:151-159, 1933. (*d*) Krombach, K.: Beitrag zur Spirocidbehandlung der kongenitalen Säuglingslues, Klin. Wchnschr. **7**:1512-1513, 1928.

16. Friedman, C. F.: Acetarsone in the Treatment of Osseous Lesions of Early Congenital Syphilis, Am. J. Dis. Child. **48**:548-564 (Sept.) 1934.

17. Pillsbury, D. M., and Perlman, H. H.: The Treatment of Prenatal Syphilis with Acetarsone (Stovarsol): Preliminary Report of Results in Seventy-Three Cases, Pennsylvania M. J. **38**:327-331, 1935.

18. Yampolsky, J.: Acetarsone in the Treatment of Syphilis in Negro Children, Am. J. Dis. Child. **48**:81-100 (July) 1934.

19. Bratusch-Marrain, A.: Method and Value of Spirocid Treatment of Syphilis in Childhood, Arch. f. Kinderh. **92**:26-39, 1931.

on the patient's weight are given. Neither system has any adequate background of animal experiments, and both were originally advanced on comparatively restricted clinical data. Much larger doses have been used by other clinicians, but data presented later indicate, we believe, that larger doses are unwise.

While the system of Maxwell and Glaser predicates the administration of slightly over three courses of acetarsone yearly, the average received by our patients was not over two. The patients were given a supply of acetarsone sufficient for one week at each visit. Weekly urinalysis was required.

Blood for a Kolmer-Wassermann and for a Kahn test was taken at the beginning and end of each course of treatment. In general, when a completely negative reaction was obtained one more course of acetarsone was given and treatment then discontinued provided no evidence of clinical or serologic relapse was noted.

TABLE 1.—*Comparative Schedules of Dosage of Acetarsone*

Maxwell and Glaser			Bratusch-Marrain		
Period of Treatment, Weeks	Dose in Tablets (0.25 Gm. Each)	Times Daily	Period of Treatment, Weeks	Dose, Mg. per Kg. of Body Weight	Times Daily
1	¼	1	1	5	1
1	¼	2	1	10	1
1	¼	3	1	15	1
1	¼	4	6	20	1
1	½	3			
1	½	4			
1	1	2			
Total: 14 Gm. in 49 days Rest period, 6 weeks			Total: Approximately 1 Gm. per Kg. in 63 days Rest period, 4 to 6 weeks		

Some selection of the patients for acetarsone therapy was exercised. It had previously been our policy when using standard measures not to employ arsphenamine initially for weak premature infants, for children severely ill as a result of syphilis or for patients suffering from an active syphilitic lesion of a vital organ. This is, of course, simply the application of a well established rule for the treatment of adult syphilis. One of us (D. M. P.) has seen 2 toxic syphilitic infants die promptly as a result of the too early use of arsphenamine. We have followed a cautious policy in the initial use of acetarsone for such patients.

When a child was started on acetarsone therapy we have, as consistently as possible in the best interests of the patient, given the drug alone. While it would theoretically be of advantage to alternate acetarsone with a heavy metal, we did not see how any accurate evaluation of acetarsone itself could be made in this way.

CLINICAL EFFECTS

The disappearance of spirochetes from cutaneous lesions has been followed daily in only 3 cases. No spirochetes were found at the end of

seventy-two, ninety-six and one hundred and twenty hours, respectively. In general, cutaneous lesions required between three and four weeks of treatment before complete disappearance occurred. The lowest recorded time for complete involution was two weeks. In a case in which acetarsone was discontinued after one week because of vomiting, relapse of the cutaneous lesions occurred. In another case a syphilid appeared for the first time after two weeks of treatment. Further treatment with the usual increasing dose caused involution, and the ultimate clinical outcome was entirely satisfactory.

In general, we should regard the effect of acetarsone on infectious lesions as distinctly slower than that of arsphenamine and certainly no more rapid than that of an insoluble bismuth salt. Wright [20] noted disappearance of cutaneous lesions in 3 cases forty-eight hours after an injection of a bismuth preparation had been given.

The satisfactory disappearance of osseous lesions as shown by roentgen examination after one course of treatment in 11 cases has been recorded in a previous paper.[17] In 2 cases of Clutton's joints involution was satisfactory.

The effect of acetarsone on interstitial keratitis in our series has been disappointing. The response of this lesion to any form of treatment is capricious, and our series was small. However, in 5 cases the course of the active lesion was prolonged, and involvement of the other eye was not prevented. In 3 cases marked corneal nebulas and significant decrease in vision were noted as the end result.

We have encountered no case of late clinical relapse after acetarsone was given. Clinical progression of the disease in spite of treatment has occurred in 8 cases.

One death occurred in a premature infant one month after acetarsone had been discontinued because of acute hemorrhagic nephritis which cleared up in one week. The patient was removed from the hospital against the resident physician's advice. We are unable to state positively whether the administration of acetarsone contributed to the child's death. We believe that it did not.

EFFECT OF ACETARSONE ALONE ON WASSERMANN AND KAHN REACTIONS OF BLOOD

In table 2 is summarized the effect of acetarsone therapy alone on the Wassermann and the Kahn reaction of the blood. The results have been set down in relation to age and amount of treatment given. Serologic negativity was not considered to have been obtained until the reaction to both antigens was completely negative. It was an almost invariable rule that when the time for serologic reversal was prolonged, the

20. Wright, C. S.: Bismuth in the Treatment of Congenital Syphilis, J A. M. A. **89**:424-428 (Aug. 6) 1927.

Wassermann (Kolmer) reaction became negative before the Kahn reaction did so. This was not true for many children under 1 year of age, in whom complete serologic reversal frequently occurs after one course of treatment.

It is noted that among children under 6 months the percentage of serologic reversal was 70. In Smith's [14] series of patients treated with arsphenamine and heavy metal the incidence of serologic reversal when treatment was begun before the age of 6 months and given regularly during the first year was 94.2 per cent. If treatment was intermittent, the percentage was reduced to approximately 80, and if very irregular, to 70. On the basis of our relatively small number of patients in this age range, the percentage of serologic reversal is lower after acetarsone therapy than after regular intensive treatment with arsphenamine and a bismuth preparation. The number of serologic reversals would prob-

TABLE 2.—*Effect of Acetarsone Alone on Wassermann and Kahn Reactions of Blood*

	Number of Cases	Final Wassermann and Kahn Tests	
		Positive	Negative
Patient under 6 months of age			
1 to 3 courses of acetarsone	20	5	15
4 or more courses	10	4	6
Totals	30	9 (30%)	21 (70%)
Patient over 6 months of age			
1 to 3 courses of acetarsone	62	44	18
4 or more courses	43	23	20
Totals	105	67 (64%)	38 (36%)

ably have been increased by more prolonged treatment in the 5 cases in which fewer than three courses of acetarsone were given, but the percentage would even then not have equaled that obtained by Smith. Whipple and Dunham [4] have collected the various reports of the effect of standard treatment on infants and found that the incidence of clinical and serologic cure varies from 59 to 97 per cent. They have also stated that although the use of sulfarsphenamine has received the unqualified endorsement of the Cooperative Clinical Group,[21] reports of the results of its use are few and not entirely satisfactory.

In the age group over 6 months a strikingly lower percentage of serologic reversal was noted—only 36 per cent as compared to the 70 per cent reversal observed among infants under 6 months. This is in accord with the reports of many observers of cases in which standard methods were used. Smith found the percentage of serologic reversal among patients over 2 years of age to be only 29 per cent.

21. Moore, J. E., and others: Management of Syphilis in General Practice, to be published.

To 47 of our patients arsphenamine and a bismuth or mercury compound were also given. In most of the cases administration of acetarsone followed the other types of therapy. Of 7 patients for whom treatment was started before the age of 6 months, all became seronegative. Of those who received a large amount (over twenty injections) of arsphenamine and a bismuth preparation, there were a considerable number who had given evidence of fixed seropositivity before we began our study of acetarsone (table 3).

Of the 196 patients who received some acetarsone, 21 had some type of reaction, an incidence of 10.7 per cent (table 4).

TABLE 3.—*Effect of Combined "Standard" and Acetarsone Therapy on Wassermann and Kahn Reactions of Blood*

	Number of Cases	Final Wassermann and Kahn Tests	
		Positive	Negative
Little arsphenamine or bismuth medication plus acetarsone	20	15	5 (25%)
Much* arsphenamine or bismuth medication plus acetarsone	27	13	14 (52%)
Total	47	28	19 (40%)
Same group tabulated as to age			
Under 6 months when treated	7	0	7 (100%)
Over 6 months	40	28	12 (30%)
	47	28	19

* "Much" arsphenamine or bismuth medication indicates over twenty injections.

TABLE 4.—*Reactions to Acetarsone*

Total number of patients receiving drug		196
Total reactions		21 (10.7%)
Nephritis	8	
Gastrointestinal disturbance	7	
Mild dermatitis	3	
Gastrointestinal disturbance and dermatitis	1	
Fixed eruption	1	
Severe peripheral neuritis, hepatitis and dermatitis	1	
Number of reactions causing permanent discontinuance of acetarsone therapy		10

REACTIONS TO ACETARSONE

We regarded all the nephritic reactions in which the presence of blood cells, casts and significantly increased albumin in the urine was the basis of diagnosis as sufficient contraindication to further attempts at acetarsone therapy. This view is not shared by some observers.[3d] The dermatitic reactions were all mild and not of sufficient severity to interdict therapy.

The most severe reaction occurred in a Negress aged 1 year, who had generalized dermatitis with a temperature of 104 F. after four weeks of treatment. During this time the scheduled dose was 4.2 Gm. Under the Bratusch-Marrain schedule the directed dose would have been 2.6 Gm. The patient was admitted to the hospital; marked hepatic enlargement was soon noted, and paralysis of the left external rectus muscle,

absence of knee and ankle jerks and bilateral foot and wrist drop developed. Definite improvement occurred within two weeks.

The number of reactions occurring in patients treated in the hospital and as outpatients revealed an interesting difference (table 5). We desired information on this point primarily to determine whether or not the prescribed drug was administered as directed to outpatients. It was known that patients treated in the ward received full, accurate doses; we had and still have doubt that such was the case when treatment was administered by the parents. It was found that the incidence of reaction in the hospital was almost four times that in the clinic (31.8 per cent as compared to 8 per cent). We were unable to explain this on the basis of any other illnesses from which the ward patients might be suffering or of the slightly lower age level of these children.

EXAMINATIONS OF SPINAL FLUID

Examination of the spinal fluid of 102 patients were made, the standard procedures of cell count, globulin determination, Wassermann test

TABLE 5.—*Incidence of Reactions in Patients Treated in Wards (Drug Known to be Administered) and in Outpatients (Administration Uncertain)*

Patients receiving acetarsone in ward	22
Number of reactions	7 (31.8%)
Patients receiving acetarsone at home	174
Number of reactions	14 (8%)

and colloidal gold test being carried out. The spinal fluid of 2 of these patients was abnormal, that of 1 showing the "paretic" formula (characteristic of dementia paralytica), with no clinical signs, and that of the other a tabetic curve, with definite clinical evidence of juvenile tabes. In 2 other patients signs of neurosyphilis were present. One of these was reported to have had a positive Wassermann reaction of the spinal fluid previously; the other had not been tested. Of the total of 187 patients with congenital syphilis, therefore, 4 had undoubted evidence of neurosyphilis, but data on the spinal fluid are not available for 84 of these. Excluding their cases, we find that the incidence of neurosyphilis in our series was slightly under 4 per cent.

DETERMINATION OF ARSENIC IN URINE

In an effort to determine the value of routine qualitative tests for arsenic as evidence of whether the patient was receiving acetarsone, a large number of tests by Gutzeit's and by Reinsch's method were done. Occasional positive reactions were obtained, but the procedures proved of no clinical value. Negative reactions were reported for most of the patients definitely known to be receiving acetarsone. No determinations on specimens of the stool were done.

COMPARATIVE ATTENDANCE OF PATIENTS RECEIVING INJECTION THERAPY AND THOSE RECEIVING ACETARSONE

The ratio of the actual to the expected attendance of patients at the clinic, whether for treatment or reexamination, was determined for the year 1930, when all patients were receiving treatment by injection. This was compared with the reports of the social service department, submitted monthly during the past three years, and with a survey made during the first year acetarsone was used. The attendance of patients given injection therapy was 51 per cent of that expected. During the first year of acetarsone therapy it was 69 per cent, but during the past three years the average has been 51 per cent, with a monthly variation of 33 to 74 per cent. The attendance ratio always fell during the summer months (average, 40 per cent) and during inclement weather (33 per cent in January 1937). Possibly the initial high attendance was incident to the enthusiasm of the staff and the social service department over a

TABLE 6.—*Basis of Diagnosis in Cases of Syphilis Recognized Within One Month after Entrance into Hospital*

Total number of cases in which syphilis was diagnosed without delay	100
Diagnosed by*	
Routine serologic study	60
Cutaneous lesions	19
Snuffles	6
Roentgenograms of bones	9
Changes in the central nervous system	4
Gross lesions of bones	2
Interstitial keratitis	3
Pseudoparalysis of Parrot, acquired syphilis, facies, Clutton's joints	1 each

* Four of the patients presented two obvious lesions.

new form of treatment or to fear of its possible dangers. At that time, also, the drug was being supplied free by the manufacturer, but some fall in attendance was noted while this was still the case. In any event, the percentage attendance of patients receiving injection therapy and acetarsone, respectively, has been exactly the same, 51 per cent.

LAG IN THE DIAGNOSIS OF SYPHILIS IN PEDIATRIC PRACTICE

As has been stated, of our series of 187 cases of congenital syphilis the disease was not recognized in 87 after the initial examination in the outpatient department. The average delay in arriving at the diagnosis in these cases was three years. The extreme occurred in a patient admitted to the outpatient department at the age of 1 month; the diagnosis of congenital syphilis was not made until the end of the pediatric age period, 12 years.

In table 6 we have summarized the essential data on the cases in which the diagnosis of syphilis was made within one month after admission to the clinic. Of 100 cases, the diagnosis was arrived at on the basis of a serologic test in 60. In 40 per cent some frank lesion of

syphilis was present. With the exception of changes in the bones, determinable only by roentgen study, the lesions due to syphilis were so prominent that even a cursory examination was sufficient to reveal them. The incidence of latency, 60 per cent, is slightly higher than in an earlier, unpublished survey by Perlman [22] of cases from our clinic, in which he found it to be 50 per cent.

The data on cases in which the diagnosis was not made within one month after registration are summarized in tables 7 and 8. It is noted that of 40 patients admitted to the clinic before the age of 1 year the diagnosis was not made before the first birthday for 25. These patients had been deprived of the major portion of their chance for cure, as the effectiveness of any system of treatment is greatly lowered after the

TABLE 7.—*Lag in Diagnosis in Clinical Pediatric Syphilology*

Number of cases in which diagnosis was delayed	87
Average time until syphilis was detected	3 years
Number of patients under the age of 1 year when registered	40
Number of patients over the age of 1 year when registered	47

TABLE 8.—*Active Congenital Syphilis Developing Under Medical Supervision*

Total number of cases	87
Total years of untreated congenital syphilis	265.7
Cases in which diagnosis was made by serologic study	65
Number of patients in whom lesions of syphilis developed after entrance into clinic	22
Types of lesions	
Cutaneous	5
Osseous	
Roentgen changes alone	5
Gross changes	2
Snuffles	2
Persistent lymphadenopathy	2
Interstitial keratitis	2
Chorioretinitis, Clutton's joints, Hutchinson's incisors, paroxysmal hemoglobinuria	1 each

age of 1 year. In table 8 it is seen that 22 of 87 patients had frank or highly suggestive lesions of syphilis during an average period of three years before the diagnosis was made. In 4 cases permanent and irremediable disability resulted in large part from failure to make the diagnosis of syphilis at the time of admission.

A review of the records in these cases showed that for all but a few patients the initial physical examination had been adequate. In 4 cases there was suggestive evidence of syphilis, but the original findings in this regard were apparently disregarded. A considerable number of records had the notation "Return for Wassermann"; the patient did not return as directed. It was apparent that in well over 90 per cent of cases the diagnosis was not established because of the physician's failure to do a routine Wassermann test of the blood on the first visit.

22. Perlman, H. H.: Unpublished data.

In only 1 case in which the initial Wassermann reaction was negative was evidence of the disease noted subsequently.

Our material furnished evidence that the diagnosis of congenital syphilis will be missed in at least 6 of 10 cases unless a serologic test is performed. Also, after the doubtful period of early infancy is passed, the serologic evidence becomes certain, and in few cases will the diagnosis fail to be made when modern antigens are used in the serologic study.

COMMENT

It has been shown that the data concerning the toxicity of acetarsone for animals are conflicting and that animal experiments cannot be used as a basis for establishing the nontoxic dose for man or for determining the spirocheticidal power and toxicity of various lots of the drug produced commercially. This is in marked contrast to the control which is exercisable in the case of arsphenamine compounds. It is possible that marked differences in tolerance exist in different species, and that the toxicity of the drug for rabbits is not a final argument against its use for man.

Some confusion concerning the necessary and safe dose for man exists. It is probable that the system suggested by Bratusch-Marrain, which is most widely used at present, is the best available, since it affords enough acetarsone to be effective on clinical lesions of syphilis, and the initial doses are low and based on the weight of the patient. That this will not completely obviate reactions, however, is certain from the recorded experience, and we have seen 7 reactions in which the dose, had it been based on weight, would have exceeded those prescribed. It is possible that smaller doses will suffice for antisyphilitic effect, and we believe that the question should have further study.

Our experience would indicate that most reactions to acetarsone are due to the pure toxic effect and not to any allergic sensitivity as in the case of an exfoliative dermatitis following use of arsphenamine. We have not performed any cutaneous tests. In the majority of cases the drug may be administered after a reaction without adverse effect. When a severe reaction has developed, e. g., nephritis, neuritis or hepatitis, we have not felt justified in resuming acetarsone therapy. While it has been stated that a nephritic reaction is no contraindication to further acetarsone therapy after the urine has become normal, we do not believe that one is justified in assuming that no change of possible future significance to the patient has occurred.

We are not convinced that even the smaller doses employed by many observers are not unnecessarily large. This is indicated by the good clinical results we have seen in patients whose attendance was irregular and to whom the prescribed doses were probably not given faithfully at home. All in all, we cannot escape the feeling that, without a signifi-

cant series of cases in which treatment throughout is under controlled conditions, with absolute assurance that the doses of acetarsone are administered as directed, figures concerning the proper dose and toxicity for human beings must be regarded as tentative and subject to question.

The much higher incidence of reaction among patients treated in the wards of the hospital, four times that observed among outpatients, seems a strong indication that treatment in the home is not carried out as directed. Our social service workers emphasized the necessity of regular treatment during visits to the patient's home, but this does not constitute anything approaching full control.

The lessened difficulty of conducting a clinic for the treatment of congenital syphilis when acetarsone is used is considerable. Venipuncture is necessary only at intervals of weeks, and the physician's calm need be interrupted only by questioning of the mother and examination of the patient for reactions. We cannot but feel, however, that a specter is always present—that the entrusting of treatment to a prescription blank may shift the responsibility for treatment of an entirely helpless and dependent child to the hands of an irresponsible parent or guardian. It is true that one can sense the irresponsible parent quickly, and we believe that he is an absolute contraindication to further acetarsone therapy. The child with congenital syphilis has already paid too great a price for the irresponsibility of one of his parents; no more should be expected of him.

From the clinical results obtained it is evident that a majority of parents realize the necessity of cooperation. The effect of the drug on evident gross lesions of syphilis is good. Acetarsone has a definitely less rapid action than arsphenamine on cutaneous lesions and possibly approaches that of an insoluble bismuth compound. It does not remove the hazard of infectiousness in an infant with congenital syphilis as rapidly as does arsphenamine or a soluble bismuth compound. The percentage of serologic reversals among patients whose treatment was started before 6 months of age was less than that reported by Smith [14] in the most adequately studied series available. However, further treatment in some of our cases and observation for a longer period may reduce the number of seropositive patients in this age group. The results in this regard are better than in some series in which standard preparations were used. The burden of proof would seem to rest on the collection of larger series of cases followed for longer periods after acetarsone therapy. That the percentage of Wassermann-fastness is significant from the standpoint of relapsing lesions in patients with early congenital syphilis is well shown by Smith's material.

In cases of late congenital syphilis (in patients over 2 years of age) the percentage of Wassermann-fastness, 67, was less than that reported by Smith. In the series of Cole and his associates [12] 53.9 per cent of

patients presenting latent syphilis on admission showed a fixed positive Wassermann reaction of the blood. Those authors showed that in the presence of active late syphilitic lesions the presence of a fixed positive reaction of the blood does not justify as good a prognosis in regard to the ultimate clinical outcome as does a more labile reaction. In late latent congenital syphilis their findings indicate that seropositivity has no bearing on the final clinical outcome, provided adequate treatment is given.

We have encountered 8 cases (5 of interstitial keratitis and 3 of neurosyphilis) in which the outcome thus far has been unsatisfactory. Two cutaneous relapses were noted in cases of early syphilis. Only 1 late serologic relapse has been seen. In cases of latent syphilis and in cases in which the initial response to acetarsone was good, not a single serious clinical relapse has occurred. This is in marked contrast to the results observed in the group of 87 cases in which no treatment was given and in which 22 clinical relapses occurred during an average period of three years.

The high proportion of latency in our cases is sufficient indication of the complete inadequacy of physical examination as a means of ruling out congenital syphilis. Routine serologic tests are carried out in the pediatric department in comparatively few hospitals; they are an absolute essential to an adequate study. They are particularly important for infants, in whom the cure of syphilis almost always can be accomplished. For newborn infants roentgen studies are also essential.[23] It is true that venipuncture in infants may be difficult and time consuming, but its performance, according to our experience, will make the prolonged treatment and observation of late congenital syphilis unnecessary in most cases. Conflicting serologic findings will undoubtedly be encountered, but adequate collateral study, particularly roentgen study of the long bones, will ordinarily establish the presence or absence of syphilis. The problem of serologic interpretation in the treatment of newborn children has been well discussed by Ingraham [24] and by Faber and Black.[25] The latter writers have shown in a small series of cases that reagin in the blood of newborn infants is in some cases simply carried over from the maternal circulation and is not evidence of syphilitic infection of the infant. In cases of syphilis with a positive Wassermann reaction active syphilis is indicated by an increase in reagin. The absence of

23. Parmelee, A. H., and Halpern, L. J.: The Diagnosis of Congenital Syphilis, J. A. M. A. **105**:563-566 (Aug. 24) 1935.

24. Ingraham, N. R., Jr.: The Diagnosis of Infantile Congenital Syphilis During the Period of Doubt, Am. J. Syph. & Neurol. **19**:547-580, 1935.

25. Faber, H. K., and Black, W. C.: Quantitative Wassermann Tests in the Diagnosis of Congenital Syphilis: The Clinical Importance of Fildes' Law, Am. J. Dis. Child. **51**:1257-1267 (June) 1936.

syphilis will be shown by a decrease in reagin, with spontaneous reversion of the Wassermann reaction to negative.

SUMMARY AND CONCLUSIONS

1. Acetarsone by mouth is an active antisyphilitic agent.

2. Its action is less rapid than that of arsphenamine.

3. No adequate experimental background for determination of the toxicity and spirocheticidal effect of individual lots of acetarsone is available.

4. In our cases the effect of acetarsone alone in reversing the Wassermann and the Kahn reaction of the blood has been only moderately satisfactory for infants under 6 months of age but as good as that of standard preparations, or better, for patients over this age.

5. The incidence of all reactions has been 10.7 per cent; that of serious reactions, particularly nephritis, 4.6 per cent.

6. For newborn infants the use of a system of dosage based on weight is essential; reactions are reduced but not entirely prevented by this means. The system of Bratusch-Marrain seems the best available.

7. Nephritic reactions occurring suddenly and insidiously are the greatest single drawback to acetarsone therapy. We do not believe that administration of the drug should be resumed after such a reaction. In general, the occurrence of gastrointestinal and dermatitic reactions does not contraindicate further acetarsone therapy.

8. Acetarsone is probably not administered as directed to patients treated at home. The incidence of reactions among patients treated under controlled conditions in the hospital was four times that observed among outpatients.

9. Among 87 cases observed for over three years after acetarsone therapy, 1 instance of serologic relapse of the blood was noted. No instance of clinical relapse of a lesion not present when treatment was started has been observed.

10. Of 87 cases under medical observation for an average of three years before antisyphilitic treatment was given, clinical relapse occurred in 22.

11. The effect of acetarsone on interstitial keratitis has been poor.

12. Regularity of attendance has not been increased by oral therapy as compared to injection.

13. The performance of a routine serologic test is an absolute essential of an adequate pediatric study. With newborn infants the serologic data must be evaluated with extreme care, but with older infants and children a repeatedly positive serologic reaction of the blood is unquestionably of serious prognostic import unless treatment is given.

14. We believe that evaluation of the responsibility of the parent or guardian is one of the most important features of treatment by mouth and that lack of cooperation by the parent is a sufficient contraindication to acetarsone therapy. The child with congenital syphilis has already paid a sufficient price for the irresponsibility of one of his parents; no more can be expected of him.

15. The effect of acetarsone in arresting congenital syphilis is inferior to that of arsphenamine and bismuth preparations. The incidence of reactions is high; acetarsone cannot be controlled by experimental studies of spircoheticidal action and toxicity in animals, and administration to outpatients is not assured.

16. The performance of a routine Wassermann test of the blood and of a precipitation test are absolutely essential to an adequate pediatric examination.

ABSTRACT OF DISCUSSION

Dr. Harold N. Cole, Cleveland: I think that Dr. Pillsbury's contribution is one of the best that has been made to medicine before this section in some time. It is well known how much the detail man is taught to emphasize acetarsone for congenital syphilis. "It is the drug of choice, particularly for infants," "You get better results in congenital syphilis with acetarsone than with any other preparation," and so forth, ad nauseam. Certainly Dr. Pillsbury did not find all those statements to be true. There is no question that acetarsone is effective against Spirochaeta pallida; it may be useful enough even to cure syphilis clinically. However, one must always remember that one is treating the patient as well as the syphilis, and when reactions occur in about 30 per cent of cases, many of them very severe (nephritis in as many as 4 per cent), one should certainly think twice before using that preparation further.

I question whether there is a place in medicine today for acetarsone unless there is an enormous improvement in it. Too many other remedies that may be used with comparative safety will give even better results.

Dr. C. C. Dennie, Kansas City, Mo.: I have had an opportunity in the last two years to go over the entire literature on acetarsone therapy, and I have been struck by the fact that there is much variation in the reports of different investigators.

I have used acetarsone only to see what actually happened in cases of acute early congenital syphilis. On reviewing the literature I came to the conclusion that acetarsone is a dangerous drug, is not therapeutically as efficient as neoarsphenamine and has no place in the treatment of either congenital or acquired syphilis. Any drug that causes as many reactions as this one does should be discarded.

It was used, of course, because it could be administered orally.

Some foreign investigators have reported as many reactions as have been reported here today, but a number of them have reported reactions of the central nervous system and effects on the hemopoietic system.

Dr. G. V. Kulchar, San Francisco: Acetarsone, like the recently revived arsenoxide, is a drug originally discarded by Ehrlich which I think deserves further investigation. Its spirocheticidal action and its effect on lesions, one gathers from

the literature (particularly from its most enthusiastic proponent, Oppenheim), are almost as rapid as those of the arsphenamines, though its effect on the Wassermann reaction is less marked.

Except in a few instances it should not be used for early syphilis. With present means of treatment, according to the statistics of the Cooperative Clinical Group, one can expect 85 per cent or more of good results; acetarsone, therefore, has no place, except perhaps for the occasional patient who is traveling and unable to receive arsphenamine or perhaps for one who cannot tolerate other arsenicals.

The chief objection to acetarsone is that the difference between the toxic and the therapeutic dose is too small. Rosahn and Kemp have studied this experimentally with rabbits. They found that the drug in therapeutic doses was lethal. There was, however, a wide variation in susceptibility, which they were unable to interpret except perhaps on the basis of wide variation in toxicity of the drug in the same batch or perhaps of variation in the susceptibility of rabbits to the drug. It is known that the rabbit is not always a satisfactory animal for the testing of preparations to be administered orally. Kemp and Rosahn (*Am. J. Syph., Gonor. & Ven. Dis.* **20**:131, 1936) have shown that changes in the gastric secretions of rabbits sometimes introduce variable factors into the experiment.

Acetarsone seems to be able to penetrate the central nervous system. Raiziss in the United States has shown this, and there have been enthusiastic reports of the use of acetarsone in the treatment of syphilis of the central nervous system. This deserves further investigation.

The reactions following acetarsone therapy have been mild. Except for the nephritis mentioned today, they have been mostly dermatitic. The most serious reactions have been those involving the nervous system. These are fortunately rare.

Before acetarsone is discarded it certainly should be investigated further, particularly in the laboratory.

Dr. W. Ray Jones, Seattle: In Seattle an enthusiastic physician conceived the idea of protecting the health of the public by dosing prostitutes with acetarsone so that they would not disseminate syphilis. He is succeeding admirably. He put some out of business by intoxicating them with acetarsone, and they came to my clinic. Being incapacitated from the effect of the drug, they could not ply their trade; hence they are not disseminating syphilis.

Dr. Donald M. Pillsbury, Philadelphia: Practically all of the fifty or more published reports on the use of acetarsone for congenital syphilis since Fourneau revived the drug in 1921 have been favorable. However, Whipple and Dunham in their recent governmental survey of the subject (and they made this survey, I think, from the pediatric point of view) have stated that in only four of the aforementioned reports were the number of cases and the period of observation sufficient to justify any conclusions.

One finds in almost all these reports the argument that acetarsone should be used because it can be given by mouth. I believe that this is a valid argument only if the clinical results compare with those of standard medicaments. It is certainly much more pleasant to run a clinic for congenital syphilis when treatment is given by mouth. We do not think, however, that our results justify its routine use.

BLOOD URTICARIA

INCLUDING A CONTRIBUTION ON METALLERGIC GENESIS OF COLD URTICARIA

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A review of the literature reveals no report of a case of urticaria due to the ingestion of blood or blood-containing foods. A recent article considered the use of animal blood for the nutrition of the German people within the scope of the second four year plan. The investigation of the findings in the following reported case is of especial interest because not only (1) is it the first such case to be reported in the literature indicating that the use of animal blood in the diet is not without its dangers but (2) it was during the nutritional allergic state of the patient that cold urticaria developed.

REPORT OF CASE

W. L., 40 years old, a helper in a milk concern, was admitted to the hospital complaining of urticaria. He had had measles as a child, malaria in 1917 and again in 1931 and typhoid fever in 1918; he had complained of recurring pains in the joints since the spring of 1937. He had heartburn after meals and was easily excited. The attacks of urticaria began in November 1937 and usually occurred after meals. The patient received ephedrine pills and about nine injections of calcium and felt improved. On Dec. 26, 1937, urticaria developed after he ate what he knew definitely to have been blood sausage. (Blood sausage is made of pork, a large proportion of pork blood, bread and pepper.)

The patient was medium sized, muscular and well nourished, with an enlarged but painless spleen. Examination of the tonsils, roentgen study of the sinuses, teeth and chest and the Wassermann test gave negative results.

After twenty-four hours on a meat-free diet in the hospital the patient was free of symptoms. About four hours after he was given blood sausage urticaria

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From the University of Pennsylvania, Clinic of Cutaneous Medicine, director, Dr. John H. Stokes (Dr. Urbach); the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital, Executive Officer, Dr. George M. MacKee (Dr. Greenberg), and the Universitätskinderklinik der Charité, Director, Prof. G. Bessau (Dr. Linneweh).

1. Footnote deleted on proof.

involved a large part of the body. When placed on a meat-free diet he again showed objective and subjective improvement. In the following days separate additions of fish, beef, veal, pork, bread and pepper to the diet did not cause any outbreak. On January 10, three hours after he had had a lunch of roast blood sausage urticarial wheals appeared on the shoulders and forearms. After he washed his face and body with cold water that evening urticaria developed on the parts washed (the first time urticaria appeared after the use of cold water) and on those areas of skin which had shown an urticarial reaction some hours before (in other words, a flare-up). The next day, when he had not been fed blood sausage and was free of symptoms the patient washed with cold water and no urticaria resulted. Some days later, after he had eaten only roast pig blood urticaria appeared in about three quarters of an hour in the region of the chest, back and face. Washing with cold water at this time resulted in more marked urticaria at the places which came into contact with the water, as on the first occasion. The patient's stomach was washed, and in about one-half hour the urticaria disappeared. The patient left the hospital, so that further investigation was not possible.

Summary.—A case is reported of urticaria following the ingestion of blood sausage (made of pork, a large proportion of pork blood, bread and pepper). Separate investigation of the individual ingredients of the blood sausage showed that the roast pork blood was the only cause of the urticaria. Further analysis to determine which part of the blood (the protein, hemoglobin, etc.), roasted or raw, was the ultimate cause and whether other animal or human blood would have caused the same manifestations was unfortunately not possible, since the patient left the hospital. It was observed that only after the development of the nutritional allergic state did urticaria follow the use of cold water.

COMMENT

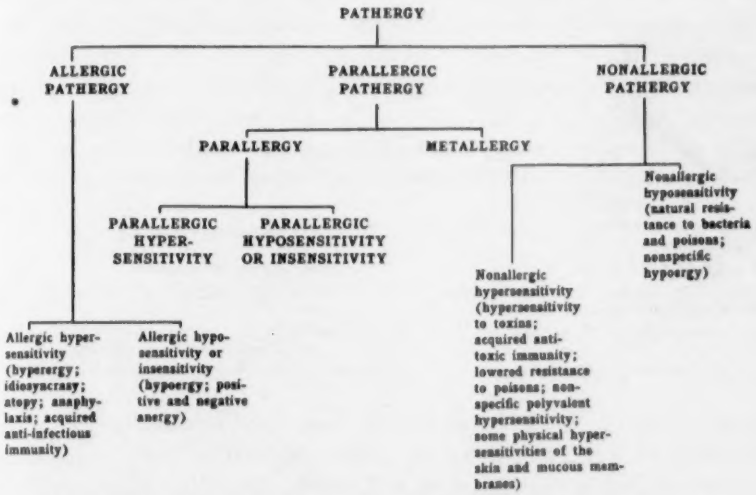
1. The first manifestation—urticaria due to the ingestion of a particular food, in this case roast pork blood—is generally understood and requires no further explanation, but the second manifestation—the development of cold allergy only during the time of an existing allergic state in the body—is of great interest and requires additional comment. We should like to suggest a possible explanation for this phenomenon, and for this purpose we must recall the conceptions of parallergy and metallergy.

By parallergy Moro and Keller[2] designated the condition of an organism which during the development of an allergy or in the time of allergic oscillations reacts in general more quickly and easily to other irritations than does a nonallergic organism. The allergic reactions caused by the second antigen are clinically different from those produced by the first. Examples are: (1) the positive tuberculin reaction at the height of vaccinia in a patient whose reaction had formerly been negative; (2) the vaccinal sore throat on the ninth or tenth day after vac-

2. Moro, E.: Monatschr. f. Kinderh. **34**:193, 1926. Keller, W.: Deutsche med. Wchnschr. **54**:307 and 345, 1928. Moro, E., and Keller, W.: Klin. Wchnschr. **14**:1, 1935.

cination; (3) the accompanying sore throat between the seventh and the ninth day after injection of serum; (4) the vaccinal encephalitis which develops nine to twelve days after vaccination; (5) the erythema nodosum of children, which appears about seven weeks after tuberculous infection, at the time of the change from the preallergic to the allergic state and (6) the phlyctenulas which develop after grip or measles.

The term metallergy was used by Urbach [3] to denote the susceptibility of an organism which has been specifically allergized to one substance to the production by other substances of the same clinical picture. Examples are: (1) the positive cutaneous reaction of a tuberculous person to the local introduction not only of tuberculin, but of horse


PATHERGY
ALLERGIC PATHERGY
PARALLERGIC PATHERGY
NONALLERGIC PATHERGY
PARALLERGY
METALLERGY
PARALLERGIC HYPER-SENSITIVITY
PARALLERGIC HYPOSENSITIVITY OR INSENSITIVITY
Nonallergic hyposensitivity (natural resistance to bacteria and poisons; nonspecific hypoergy)
Allergic hypersensitivity (hyperergy; idiosyncrasy; atopy; anaphylaxis; acquired anti-infectious immunity)
Allergic hyposensitivity or insensitivity (hypoergy; positive and negative anergy)
Nonallergic hypersensitivity (hypersensitivity to toxins; acquired antitoxic immunity; lowered resistance to poisons; nonspecific polyvalent hypersensitivity; some physical hypersensitivities of the skin and mucous membranes)

Subdivision and nomenclature of the allergic reactivities (Urbach).

serum or bouillon, (2) the flare-up of the sites of injections of old tuberculin or luetin following injections of milk, or ultraviolet irradiation, (3) the sensitivity to a hitherto unused protein of a rabbit previously given injections of three or four other proteins at five day intervals.

The basic differences between the conceptions of parallergy and metallergy are therefore as follows: 1. In parallergy there are different and in metallergy the same clinical appearances. 2. Whereas in parallergy a flare-up effect is impossible because the phenomena never occur at the place of the primary allergic reaction, in metallergy a large part of the reaction is based on a flare-up mechanism. 3. Parallergy occurs only at the time of allergic development or great fluctuation, whereas

3. Urbach, E.: Klinik und Therapie der allergischen Krankheiten, Vienna, Wilhelm Maudrich, 1935.

metallergy can accompany but does not require a momentary strong allergic reaction—in other words, is more or less independent of the allergic state momentarily present. 4. The parallergic condition can never be called forth a second time, while the metallergic state by means of the mechanism of haptenization may originate often and lead in time to a polyvalent nonspecific pathergy.

The chart gives the nomenclature and subdivisions of the allergic reactivities and may bring about a better understanding of the manner in which metallergy and parallergy fit into the system. The table presents the differences between metallergy and parallergy.

2. On the basis of the conception of "metallergic origin" this case may serve as a bridge between the controversial concepts of the pathogenesis of physical urticaria. Two groups of authors have considered the subject, those who adhere to the opinion that cold, heat, light and pressure urticaria have an allergic background and those who deny the

Differences Between Metallergy and Parallergy

Parallergy	Metallergy
Clinical manifestations different from the original reaction	Clinical manifestations the same
Never a flare-up reaction	Often a flare-up reaction
Occurs only during the development of allergy or great allergic oscillations	Occurs independent of the allergic state present
Can never be called forth a second time	Can be called forth often and leads to a polyvalent nonspecific pathergy

allergic basis of these phenomena because they are unable to transfer this kind of hypersensitivity by either the method of Prausnitz and Küstner or that of Königstein and Urbach. Our case may furnish the explanation as follows: The "cold" is not the primary allergen, but a secondary allergen induced by the first on the basis of metallergy. Every physical or chemical agent, according to the generally approved hapten theory of Landsteiner, may become a hapten if combined with a so-called conjugate protein antigen (*Schleppersubstanz*). In our case the conjugate protein antigen was the pork blood, while the hapten, secondary antigen or metantigen (according to one's choice of nomenclature) was the physical agent "cold." Thus, in a person formerly not sensitive to a physical agent ("cold") urticaria developed on the parts exposed and a flare-up reaction occurred on the areas allergized by food.

Further, from our knowledge of similar manifestations, that if a metantigen has worked often as such it may become able to work alone later, producing a so-called conditioned allergic reaction, we may conclude that at some time cold alone will be able to produce urticaria. Then, if the body is sufficiently allergized, other irritants of various physical

and chemical natures may play the role of metallergen and slowly a polyvalent nonallergic sensitivity develop. By clinical observation it will be possible often to demonstrate development from allergy to metallergy to haptenization to polyvalent nonallergic pathergy.

SUMMARY AND CONCLUSIONS

A case of urticaria following the ingestion of pork blood sausage is reported. This is believed to be the first case of urticaria following ingestion of blood described in the literature.

Should use of animal blood for nutritional purposes become widespread, more cases of intolerance should be expected.

Cold water urticaria developed during a specific food allergy. The cold water urticaria could be reproduced only after a preceding specific allergic attack.

An attempt is made to explain cold urticaria on the basis of metallergy, thus bridging the gap between the different opinions about the pathogenesis, allergic and nonallergic, of so-called physical urticaria.

Explanation of the terms parallergy and metallergy is given.

CHRONIC RELAPSING URTICARIA AND ANGIONEUROTIC EDEMA

REPORT OF A CASE WITH ASSOCIATED PANCREATIC INSUFFICIENCY AND RELIEF BY ORAL ADMINISTRATION OF DEINSULINIZED PANCREATIC EXTRACT

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I am prompted to report a case of the association of cutaneous allergy with pancreatic insufficiency, since a search of medical literature reveals little mention of this unusual syndrome.

REPORT OF CASE

G. S., a white woman aged 51, presented a generalized eruption consisting of whitish and pinkish wheals, accompanied with itching and burning. The onset was on Aug. 20, 1937, and she attributed it to her menopause. The past history revealed nothing significant except a severe attack of mumps at the age of 35. Her recovery was complicated by a peculiar "swelling sensation" across the small of the back, repeated attacks of indigestion and occasional attacks of diarrhea following ingestion of cream or a fatty meal. For the past year she has received hypodermic injections of epinephrine hydrochloride for attacks of angioneurotic edema affecting the lips, tongue and pharyngeal mucous membrane and associated with a generalized urticarial eruption. These attacks did not follow ingestion of sea food, liver, tomato or strawberries. The outbreaks did not appear to be seasonal. Her vitamin intake, including ingestion of citrus fruits,[1] was adequate. There was no history of chronic foci of infection, nor were any discernible on examination. She had never used senna as a laxative.[2]

Examination.—The patient was slender and 65 inches (165 cm.) tall and weighed 110 pounds (50 Kg.). During an attack of generalized urticaria there was swelling of the lips, tongue and pharynx, producing dysphagia and dysarthria. Examination of the head, neck, chest and abdomen showed nothing abnormal except an ocular refractive error, which was later corrected. The blood pressure was 110 systolic and 70 diastolic. A blood count, a determination of blood sugar and urinalysis gave normal results. The basal metabolic rate was + 10 per cent. The Wassermann reaction of the blood was negative. A cholecystogram showed a normal-filling gallbladder, free of infection or calculi. Cutaneous tests with wheat and milk gave positive results, although she had eaten these foods all her

1. Rosenberg, W. A.: Vitamin C Deficiency as a Cause of Urticaria, Arch. Dermat. & Syph. **37**:1010 (June) 1938.

2. Hollander, L.: Urticaria Due to Senna Leaves, J. A. M. A. **100**:1329 (April 29) 1933.

life. The stools were large, frothy and rancid and on microscopic examination showed free fat in abundance and some undigested meat fibers. There were no ova, parasites,[3] amebas or tubercle bacilli.

Course and Treatment.—From Aug. 22, 1937, until June 1, 1938, she received treatment of various kinds at different intervals, including elimination of wheat and milk from her diet, Rowe elimination diets, courses of estrone (theelin), splenic extract, calcium and nerve sedative medications and biliary drainage, only to have recurrent outbreaks of urticaria and edema of the lips, tongue and pharynx, which confined her to bed. Relief was obtained with saline catharsis, colonic irrigations and hypodermic administration of epinephrine hydrochloride. In view of the presence of abundant free fat and some protein fibers in the stool, a prolonged history of indigestion and backache following a severe attack of mumps and the occurrence of occasional attacks of diarrhea following ingestion of cream or a fatty meal, I attributed the eruption to deficient production of steapsin and trypsin enzymes, with incomplete metabolism of lipoid and protein, causing an excretion of free fat and absorption of split protein products, which produced cutaneous sensitization. Administration of deinsulinized pancreatic extract, 15 grains (0.97 Gm.) three times a day after meals, was instituted June 1, 1938, and almost complete relief of symptoms was noticed from June 15 until August 15, at which time medication with pancreatic extract was discontinued. On September 1 a fresh outbreak of hives with edema of the oral mucous membranes occurred, and she was confined to bed. Treatment with pancreatic extract was resumed and was followed by local and general relief. This medication was discontinued on November 23, and a generalized urticarial eruption appeared on November 30, which subsided in three days with oral use of pancreatic extract. Great relief of indigestion, absence of diarrhea and of steatorrhea and gain in weight were noted.

COMMENT

Deficiency of pancreatic external secretion is best attributed to a complicating pathologic disturbance of the pancreatic acini following mumps, whether it be parenchymatous degeneration, edema or actual inflammation. The cause of angioneurotic edema at times is both multiple and complex. Finding of the offending allergen and its removal from the environment may offer considerable relief. Desensitization with a specific allergen often aids. Menagh [4] described a series of cases in which there was associated infection of the biliary tract and obtained satisfactory results by administration of cholagogues and biliary drainage. Absorption of split protein products due to pancreatic disease is akin to absorption of foreign protein (allergen) from other sources and is capable of producing identical allergic reactions, such as urticaria and angioneurotic edema.[5] A history of disease of the alimentary tract, of the biliary tract and of the pancreas should be carefully searched for.

3. Endule, E.: Persistent Angioneurotic Edema, J. Allergy **3**:583 (Sept.) 1932.

4. Menagh, F. R.: Etiology and Results of Treatment in Angioneurotic Edema and Urticaria, J. A. M. A. **90**:668 (March 3) 1928.

5. Maddox, A. S.: Angioneurotic Edema, M. Rec. **11**:556 (Dec. 16) 1936.

SUMMARY

A case of chronic relapsing urticaria with angioneurotic edema associated with pancreatic insufficiency is reported. Oral substitution therapy with deinsulinized pancreatic extract caused almost total relief of local and general symptoms, which recurred on withdrawal of the medicament.

CRYOTHERAPY FOR ACNE AND ITS SCARS

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AND

CHARLES LERNER, M.D.

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The object of this paper is to report the results of a treatment for acne and its scars not previously presented in the American literature.

Solidified carbon dioxide was first used in the treatment of acne by Pusey,[1] in 1907. As early as 1909 Zeisler [2] wrote, "We have in carbon dioxide snow an exceedingly valuable agent with great further possibilities." In 1913 H. Béclère [3] employed a mixture of solid carbon dioxide and acetone in silver tubes with pressure. The year before (December 1912), Professor Bordos [4] presented before the Academy of Science of Paris a similar apparatus, which he named "cryocautery." This seems to be the origin of the term cryotherapy.

In 1925 Giraudeau,[5] at the Hôpital St. Louis, commenced using cryotherapy for acne, with a mixture of solid carbon dioxide, acetone and precipitated sulfur; in 1928 he [6] reported this therapeutic method before the French Society of Dermatology and Syphilology; this work received but passing mention, at the time, in the form of an abstract in the *Archives of Dermatology and Syphilology*.[7] A search of the literature reveals no subsequent mention of cryotherapy per se.

From The Skin and Cancer Unit, New York Post-Graduate Medical School and Hospital, Columbia University.

1. Pusey, W. A.: The Use of Carbon Dioxid Snow in the Treatment of Nevi and Other Lesions of the Skin: A Preliminary Report, J. A. M. A. **49**:1354 (Oct. 19) 1907.

2. Zeisler, J.: J. Cutan. Dis. **27**:32 (Jan.) 1909.

3. Béclère, H.: Bull. Acad. de méd., Paris **49**:455 (May 13) 1913.

4 Bordos, cited by Jeanselme and Giraudeau: Rev. gén. de clin. et de thérap. (pt. 2) **38**:657 (Oct. 11) 1924.

5. To the personal knowledge of one of us (F. L. K.) who worked with Dr. Giraudeau at the Hôpital St. Louis from 1927 to 1936.

6. Giraudeau: Bull. Soc. franç. de dermat. et syph. **35**:463 (June 14) 1928.

7. Giraudeau: Bull. Soc. franç. de dermat. et syph. **36**:654 (July) 1929; abstracted, Arch. Dermat. & Syph. **21**:308 (Feb.) 1930.

APPARATUS AND TECHNIC

The apparatus consists of a large clay mortar and pestle, solid carbon dioxide, acetone, precipitated sulfur, absorbent cotton, a square of gauze and a wooden tongue depressor.

Approximately 5 ounces (140 Gm.) of solid carbon dioxide is placed in a large clay mortar and ground to a fine powder. Acetone (dimethylketone) is carefully added, with constant stirring until a smooth paste is obtained. Then, with a wooden spatula, approximately ¼ ounce (7 Gm.) of precipitated sulfur is added, until a light yellow mixture is formed. If necessary a few additional drops of acetone may be added.

About one third of the paste is placed on a tampon of absorbent cotton, covered with a square of gauze and applied under moderate pressure to the most prominent lesions.

The tampon is then immersed a second time in the paste. At this stage the addition of a small amount of acetone is usually necessary. The carbon dioxide paste is now applied directly to the skin. The application is made without pressure, and the entire face is covered with rapid friction. The resulting deposit of sulfur is allowed to remain on the face for approximately twenty minutes after the treatment. Immediately after application, erythema and slight edema appear, which last about two or three days and are followed by mild exfoliation of the epidermis. Applications are repeated at weekly intervals.

NOTE.—Acetone is inflammable.

THEORY OF ACTION

The acetone serves a triple function: Firstly, it dissolves the sebum, permitting penetration into the follicles; secondly, it seems to increase the speed with which the solid carbon dioxide changes to the gaseous state with consequent further lowering of the temperature; lastly, it produces a more uniform paste.

Some of the sulfur is carried into the follicles, and we believe that the deposited particles exert an inhibitory action on the sebaceous secretion.

The mixture causes superficial refrigeration with subsequent exfoliation. This exfoliation is the result of an inflammatory process, with varying amounts of edema. The therapeutic response is due to the repeated exfoliations.

RESULTS IN FIFTY CASES

Over a period of eighteen months 50 patients with acne and postacne scars were treated by this method. Forty-seven of them were apparently either cured or greatly improved. This represents satisfactory results in 94 per cent of the cases. Two patients failed to respond, and 1 suffered a recurrence.

Thirty-three (66 per cent) were cured or greatly improved within four months. Another fourteen (28 per cent) were equally benefited but required somewhat more than four months of treatment.

The duration of treatment varied from a minimum of six weeks for mild conditions to a maximum of eight months for severe postacne scarring. Four months was the average time required for satisfactory results.

COMMENT

Lest the results of this method of therapy be overestimated or perhaps somewhat misunderstood, we wish to make it clear at the outset that we have not deduced definite conclusions from the 50 cases in which the treatment has thus far been used. We are fully aware that this number is far too small to carry much weight and that the results are to be viewed in the same light as those obtained with superficial roentgen therapy. Our chief aim is to acquaint the dermatologist with another useful weapon against acne and the sequelae of this frequently destructive and disfiguring disease, with the hope that as its use becomes more widely disseminated an improved technic will result.

In this series of cases a few patients that had previously been treated either insufficiently or unsuccessfully by other methods, including roentgen irradiation, were accepted for the therapy, together with those presenting themselves for treatment for the first time in the course of the disease. All patients were given the advice which is routine in cases of acne concerning such matters as diet, general hygienic measures, avoidance of bromides and the use of iodized salt. In the cases in which the condition proved resistant to cryotherapy it had been resistant to roentgen therapy also. A possible explanation for this subbornness may be the depth of many of the lesions. However, we observed some cases also in which the condition cleared with this form of treatment although roentgen rays had failed previously. Moreover, patients who had been cured by roentgen therapy showed further cosmetic improvement when cryotherapy was used solely with the aim of rendering the postacne scars and pits less conspicuous. The improvement in these cases may be explained, in all probability, by the fact that the margins of the pits and scars were diminished in sharpness of outline and that circulation was enhanced, with resulting increase in the tonus of the skin.

Our impression at present leads us to believe that cryotherapy deserves particular mention in the local treatment of acne of puberty and prepuberty. Likewise, it has proved to be a useful therapeutic agent for patients who have received maximum roentgen therapy without sufficient clinical improvement.

As is the case with other methods of therapy, one must bear in mind that there may be definite contraindications, and not the least important among them we consider sequelae of irradiation, no matter how mild. Cryotherapy should be guardedly administered when there is atrophy of the skin even when a history of roentgen irradiation cannot

be elicited from the patient. In our opinion another contraindication, perhaps more important than radiodermatitis, is the presence of a melanotic nevus within the affected area or in its immediate vicinity.

Perhaps the most important admonition that we should mention is that one should not seek to set up more than a superficial inflammatory reaction. In our opinion, it is this clinically observed inflammatory process—unfortunately not yet studied histologically—that produces the desired result. We warn against overzealousness of efforts to obtain exfoliation in the treatment of the scars of acne. Mild rather than severe exfoliation should be considered the better choice. We refrained from repeating the process of exfoliation before complete subsidence of the preceding treatment. Cures are reported with considerable reservation, because no case has been observed for a sufficient length of time to warrant considering the cure permanent.

SUMMARY

Fifty patients with acne or postacne scars were treated with a mixture of solid carbon dioxide, acetone and precipitated sulfur.

The technic is described in detail.

Over 90 per cent of the patients were apparently cured or much improved within four months.

In our opinion this form of therapy is particularly valuable for the acne of puberty.

This treatment deserves due consideration in the management of cases in which no response has followed maximum roentgen therapy.

The term "cryotherapy" is used.

SYPHILITIC REAGIN IN BLOOD AND IN SPINAL FLUID

A COMPARATIVE QUANTITATIVE STUDY

ALEXANDER S. WIENER, M.D.
AND
IRVING M. DERBY, M.D.
BROOKLYN

The purpose of the study reported here was to compare the amount of syphilitic reagin in the serum and in the spinal fluid of patients with neurosyphilis. A number of such studies have already been made, but the results have been inconsistent.[1] Whereas, to our best knowledge, all the previous work was done with some variation of the Wassermann technic, one of the flocculation technics, because of their greater simplicity, particularly for quantitative studies, was selected for use in the present investigation. A method which seemed especially suitable for the purpose, because of the clarity of the reactions as well as the relatively small amounts of serum and spinal fluid required, was the Kline test.

The present study differs from previous reported ones also in the greater size of the series studied, namely, a total of 1,245 pairs of serum and spinal fluid. All the specimens tested came from patients at the Brooklyn State Hospital for nervous and mental diseases suspected of having syphilis of the central nervous system.

The subsequent clinical course and the results of the serologic tests demonstrated that a certain number of the patients were actually nonsyphilitic, and there were many with dementia paralytica or the tabetic

Aided by a grant from the Committee on Scientific Research of the American Medical Association.

From the Office of the Chief Medical Examiner of New York City, and the Laboratories, Brooklyn State Hospital.

1. (*a*) Katzenelbogen, S.: Cerebrospinal Fluid and Its Relation to the Blood, Baltimore, Johns Hopkins Press, 1935. (*b*) Dujardin, B.: L'index de permeabilité, Ann. de mal. vén. **79**:129, 1920. (*c*) Kafka, V.: Atypische serologische Befunde bei Paralyse und ihre Bedeutung, Ztschr. f. d. ges. Neurol. u. Psychiat. **56**:260, 1920. (*d*) Auguste, C., and Taboureich, L.: Comparaison du taux des réagines syphilitiques dans le sérum et le liquide céphalorachidien, Compt. rend. Soc. de biol. **127**:1314, 1938. (*e*) Eagle, H.: Laboratory Diagnosis of Syphilis, St. Louis, C. V. Mosby Company, 1937, p. 329. (This reference contains a complete bibliography.)

form of dementia paralytica whose serologic reactions had become negative under treatment. Patients with cerebrovascular syphilis made up the vast majority of those with a positive reaction of the blood and a negative reaction of the spinal fluid, whereas almost all the patients whose spinal fluid gave a positive reaction were suffering from dementia paralytica or the tabetic form of dementia paralytica. There were only a few instances of congenital syphilis, latent syphilis or simple tabes.

With all specimens the actual tests, both qualitative and quantitative, were performed by one of us (I. M. D.); but in a number of instances, some of which will be described later, tests were carried out by both of us independently.

TECHNIC

When any new patient is admitted to the Brooklyn State Hospital, blood is drawn for a serologic examination. If there are symptoms or signs pointing to syphilis, spinal fluid is drawn at the same time. When there are no such symptoms, if the Wassermann and/or the Kline reaction of the blood prove to be positive, a spinal tap is made on receipt of the report. In the present study, the interval between the examination of the blood and that of the spinal fluid never exceeded two weeks, and in the great majority of cases both specimens were taken simultaneously. Since it is customary in cases of syphilis to repeat the examination at intervals of six months, our findings on 1,245 pairs of specimens of serum and spinal fluid represent tests on a somewhat smaller number of patients.

The serums were separated in the usual manner and then inactivated in a water bath at 56 C. for thirty minutes. The vast majority of the spinal fluids were clear and tested directly. Grossly bloody spinal fluids were not used. In the case of microscopically bloody fluids, the cells were removed by centrifuging before the tests were made. All the spinal fluids were tested unheated, since preliminary tests had shown that the titers in the flocculation test were the same with unheated fluids and those heated for periods up to at least an hour.

Each serum and spinal fluid was subjected to preliminary qualitative tests before titrations were carried out. These were the so-called diagnostic and exclusion tests for serum and spinal fluid, carried out in accordance with the original technic described by Kline.[2] It might be mentioned here that the qualitative tests for serum and spinal fluid are carried out differently.

When the qualitative tests were positive the fluids were titrated to ascertain the reagin content.[3] In order that the results of the tests on serums and spinal fluids might be comparable, the same technic was used for both. The test antigen used was the so-called Kline exclusion emulsion for tests on heated serums.

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2. Kline, B. S.: Microscopic Slide Precipitation Tests for Syphilis, Baltimore, Williams & Wilkins Company, 1932.

3. For a full description of the quantitative flocculation test using the Kline antigen see A. S. Wiener's article "Studies on the Kline Test for Syphilis," J. Lab. & Clin. Med. **22**:1062, 1937.

Progressively doubled dilutions of each serum and spinal fluid were prepared, and a drop (0.05 cc.) of each dilution was transferred to successive wells of paraffin-ringed slides. A small drop (0.008 cc.) of the test emulsion was then added, the slides rotated for four minutes and the reactions read. The reciprocal of the highest dilution of the serum still having a distinct reaction was taken as equal to the number of units of syphilitic reagin present in the serum.

In order to ascertain the limits of accuracy and reproducibility of the quantitative flocculation procedure just described, a number of the specimens were divided and tested independently by both of us in our respective laboratories. In table 1 are given the results of such an experiment. It will be noted that on the whole the agreement was satisfactory. The results would undoubtedly have been still better had it not been for the circumstances under which the tests were carried out. Thus, the specimens were all examined as a matter of routine and in large numbers at one time. For example, frequently as many as 75 serums and 25 spinal fluids were tested in one day, and of these as many as one fifth of the serums

TABLE 1.—*Comparison of the Results of Duplicate Quantitative Kline Tests Carried out Independently* to Ascertain Reproducibility*

	Number of Units of Reagin in			
	Blood Serum		Spinal Fluid	
Specimen	D.	W.	D.	W.
1	256+	256+	2	1
2	32	16+	8	4
3	0	0	0	0
4	16	16+	0	0
5	128	128	8	8
6	128	128+	4	4
7	32	32	2	2
8	2	2	2	2
9	256	128	4	4
10	2	2	2	2

* By each of us.

and half of the spinal fluids gave positive reactions and had to be titrated. Therefore, an occasional small discrepancy, such as that obtained for the spinal fluid specimen 2, is not remarkable. Such apparent differences in the results could be eliminated by repetition of the experiments. Therefore, the method selected for the present study gives readily reproducible results, having a limit of error, as a rule, of no more than one dilution.

RESULTS

In table 2 are listed the results of the tests made in our series by the quantitative technic just described. Each pair of specimens has been classified according to reagin content. Specimens which gave no reaction are classified under the heading "0." Those giving only a doubtful reaction or showing not more than 1 unit of reagin were placed under the heading "0-1." Serums and spinal fluids containing more than 1 and up to and including 2 units of reagin were placed under the heading "1-2." Those of higher titers were classified in a similar manner.

Table 2 presents several interesting features. In the first place, whereas the reagin content of the serum exceeded 64 units in about 5 per cent of the cases and in several instances reached as high as, or even exceeded, 500 units, in no instance did the reagin content of the spinal fluid exceed 16 units. Because of the rarity of cases in which the titer of the reagin was higher in the spinal fluid than in the blood, it seemed of importance to subject them to special scrutiny. Therefore, whenever possible, the tests were repeated, and in practically every instance the original findings were confirmed. During the first half of the studies such tests were not repeated, because their exceptional nature was not recognized at the time. Unfortunately, the most outstanding exception, namely, the one designated by parentheses in table 2, in which

TABLE 2.—*Comparison of Quantities of Syphilitic Reagin in Serum and Spinal Fluid in a Series of 1,245 Pairs of Specimens*

Amount of Syphilitic Reagin (Units) in Spinal Fluid*	Amount of Syphilitic Reagin (Units) in Serum*									
	0	0-1	1-2	2-4	4-8	8-16	16-32	32-64	Over 64	Totals
0	222	99	94	76	96	68	54	23	16	748
0-1		17	12	24	29	32	24	12	4	154
1-2	1	6	9	10	35	30	41	14	10	156
2-4			3	6	12	14	34	27	13	109
4-8				1	4	4	12	22	14	57
8-16				(1)†		2	6	6	6	21
Totals	223	122	118	118	176	150	171	104	63	1,245

* The inclusive numbers used cover the second figure but not the first.

† Technical error (?).

The figures in this table represent numbers of pairs of specimens. For example, there were 222 pairs of specimens of spinal fluid and serum of which each member gave a negative reaction with Kline antigen; there were 99 pairs of which the spinal fluid gave a negative reaction and the serum gave a quantitative reaction of no more than one unit.

the titer of the spinal fluid was 16 and that of the blood only 4, occurred early in the study. Since further tests were not done, it is possible that these results arose through some technical fault, but this explanation cannot be applied to the other, less striking exceptions. Here one must take into account certain unavoidable differences in sensitivity of flocculation tests on blood and spinal fluid, even when these are carried out in an identical fashion. This difference in sensitivity is apparently due to the presence in the serum of inhibiting substances not present in the spinal fluid. This can be demonstrated by carrying out comparative titrations on syphilitic serum (or spinal fluid) with saline solution, normal spinal fluid and normal serum as diluting fluids. In such tests the titers are highest when saline solution is used as the diluent, slightly lower when normal spinal fluid is used and decidedly lower with normal serum.

A positive Wassermann reaction of the spinal fluid in the face of a negative reaction of the serum is reputed to be not uncommon in neurosyphilis.[1e] Similarly, when quantitative Wassermann tests have been made, the titer of the reagin in the spinal fluid has often been reported to be higher than that in the blood serum.[1c] From these observations certain investigators have been led to the conclusion that syphilitic reagin is often present in the spinal fluid even though absent from the blood serum, and that when reagin is present in both fluids the quantity in the spinal fluid can exceed that in the serum.

When this study was undertaken, we had more or less accepted the interpretation just given and expected our findings with the quantitative

TABLE 3.—*Comparison of Wassermann and Flocculation Technic in Cases of Positive Wassermann* Reaction of the Spinal Fluid and Negative Reaction of the Blood*

Specimen	Blood Serum		Spinal Fluid	
	Wassermann	Flocculation Titer	Wassermann	Flocculation Titer
9	Negative	4	Positive	1
38	Negative	1	Positive	0
73	Negative	0	Positive	2
134	Negative	2	Doubtful	0
{235	Negative	1	Positive	0
{556	Negative	1	Positive	0
{285	Negative	0	Positive	0
{530	Negative	0	Doubtful	0
{258	Negative	0.5	Positive	0
{591	Negative	0	Positive	0

* The Wassermann tests were performed by the New York State Department of Health Laboratories.

Whereas the Wassermann technics used were different for blood and spinal fluid, the quantitative flocculation method was the same for both. The approximate relative sensitivity of the tests was as follows: spinal fluid Wassermann test > spinal fluid flocculation test = serum flocculation test > blood Wassermann test. Specimens bracketed together were taken from the same patient at intervals of approximately six months.

Kline test to confirm it. As a matter of fact, as has just been demonstrated, despite the relatively large size of our series, there were few cases in which more reagin was demonstrable in the spinal fluid than in the serum (table 2).

The explanation for the apparent discrepancy between our results and those formerly reported can be found in the difference in the method of performing the tests. Whereas one and the same quantitative technic was used for both serum and spinal fluid in the present study, the Wassermann tests customarily performed for the two fluids are different. Thus, the dose of spinal fluid ordinarily used in the Wassermann test varies between five and ten times the quantity of serum used. In addition, there are inhibiting substances in the blood serum which diminish the sensitivity of the Wassermann test of the blood; such substances are lacking in the spinal fluid. It is evidently possible, there-

fore, for the Wassermann reaction of the spinal fluid to be positive and that of the blood negative even though there is actually more reagin in the blood than in the spinal fluid. In fact, a number of such cases were encountered in the present study, and these are given in table 3. It will be seen that in 7 cases (embracing 10 examinations) the Wassermann reaction of the spinal fluid was positive but that of the blood was negative. However, when the described quantitative flocculation technic was used, in only 1 instance did the spinal fluid react to a higher titer than the blood serum, and here one must take into account the possible effect of inhibiting substances present in the blood serum but lacking in the spinal fluid. Incidentally, the number of instances of a positive Wassermann reaction of the spinal fluid and a negative reaction of the blood was relatively small in comparison with other reports.[4] This can be explained by recent improvements in technic of the Wassermann test and the decrease in disparity between the sensitivity of the test of the spinal fluid and that of the blood, due particularly to the increase in sensitivity of the latter test.

COMMENT

The findings presented are of interest in connection with the problem of the site of origin of the reagin in the spinal fluid. Two explanations have been offered for the presence of syphilitic reagin in the spinal fluid of patients with neurosyphilis: (1) that the antibodies are formed locally, and (2) that they are derived from the blood serum by filtration through the blood-brain barrier.[1b] The main argument which has been offered in favor of the latter explanation is that rarely, if ever, is there more reagin in the spinal fluid than in the serum. Although, as we have shown in this paper, it is true that the reagin content of the spinal fluid is hardly ever greater than that of the serum, this is not a valid argument for Dujardin's theory. In a previous paper [5] we presented evidence which apparently contradicted Dujardin's concept and indicated that the syphilitic reagin in the spinal fluid of patients with neurosyphilis is at least in large part formed locally. This was done by comparing the isoagglutinin and the reagin titer in the blood and spinal fluid of such patients. Were the theory of passive filtration correct, a parallel filtration of isoagglutinins could be expected. However, in no case was any isoagglutinin demonstrable in the spinal fluid, even when the reagin titer was equal in blood and spinal fluid and the isoagglutinin

4. Katzenelbogen, S.; Rogovine, S., and Monedjikova, V.: Relation Between the Wassermann Reactions in the Blood and in the Cerebrospinal Fluid: Contribution to the Question of the Origin of Antibodies, Arch. Neurol. & Psychiat. **21**:376 (Feb.) 1929.

5. Wiener, A. S., and Derby, I. M.: Site of Origin of Syphilitic Reagin in Spinal Fluid of Patients with Neurosyphilis, Proc. Soc. Exper. Biol. & Med. **38**:487, 1938.

titer in the blood higher than that of the reagin. This must not be taken to mean that there is never any filtration of reagin from the blood into the spinal fluid, since the two explanations offered for the presence of reagin in spinal fluid are not mutually exclusive.

Since our evidence favors the theory of local formation of syphilitic reagin in the spinal fluid, it is of interest to inquire what significance is to be attached to the fact that the titer of reagin is consistently lower in the spinal fluid than in the blood. It may be that the capacity to form syphilitic reagin (and other antibodies) is less highly developed in the central nervous system than elsewhere, possibly because of its small number of reticuloendothelial cells.

The results of the present investigation should be of value to the clinician from the standpoint of diagnosing syphilis of the central nervous system. Thus, in the present series not a single case was encountered at the initial diagnostic examination in which the Kline exclusion test on serum was negative and the flocculation or Wassermann test on the corresponding spinal fluid positive; and only 3 cases were found in which after treatment the Kline exclusion test on the serum was negative but the spinal fluid gave a positive reaction (table 3). Therefore, when the question of the presence or absence of syphilitic meningoencephalitis arises, this diagnosis can be practically excluded by examination of the serum alone when a sensitive test like the Kline exclusion test gives a negative result.

SUMMARY

The concentrations of syphilitic reagin in serum and spinal fluid were compared in a series of 1,245 pairs of specimens, by a quantitative flocculation technic. It was found that with the method used titers as high as 500 were attained in the blood serum, whereas the titer of the spinal fluid never exceeded 16. Moreover, the amount of reagin in any spinal fluid was practically always less than that of the corresponding serum. The bearing of these observations on the question of the site of origin of syphilitic reagin in the spinal fluid of neurosyphilitic patients is discussed.

PATHOLOGIC FRACTURE DUE TO SYPHILIS

REPORT OF A CASE WITH BIZARRE ROENTGEN FINDINGS AND FAVORABLE RESPONSE TO THERAPY

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Dermatologists are increasingly appreciative of visceral syphilis. This report demonstrates some unusual manifestations in syphilis of the bones and viscera.

Syphilis has long been recognized as a cause of pathologic fracture. Such fractures, however, are becoming increasingly less common. The older writers emphasized syphilis, and the more recent ones have mentioned it in considering the causes of pathologic fracture. Thus Hazen [1] stated: "Fractures as a result of bone destruction are frequently seen and are so frequent as to arouse always in such cases the suspicion of syphilis." Fragenheim [2] devoted considerable discussion to syphilitic fracture and Boyd [3] admitted the possibility of such phenomena. Eliason,[4] in reviewing pathologic fractures of various causes, cited Speed as stating that large numbers of spontaneous fractures due to syphilis are seen each year at the Cook County Hospital. From what follows this citation, however, it seems probable that most of these fractures are neurogenic (tabes, dementia paralytica) rather than due to actual syphilitic involvement of bone.[5] Eliason went on to say "Syphilitic disease of bone *per se* is seldom a cause of pathological fracture." Bowman [6] listed syphilis in a paper on pathologic fracture, but

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From the Service of Dr. Elmore B. Tauber, Director of Dermatological Service, the College of Medicine, University of Cincinnati, the Cincinnati General Hospital.

1. Hazen, H. H.: Syphilis: A Treatise on Etiology, Pathology, Diagnosis, Prognosis, Prophylaxis, and Treatment, St. Louis, C. V. Mosby Company, 1919, p. 253.

2. Fragenheim, P.: Die Syphilis der Knochen, in Jadassohn, J.: Handbuch der Haut- und Geschlechtskrankheiten, Berlin, Julius Springer, 1928, pp. 201-203.

3. Boyd, W.: Surgical Pathology, Philadelphia, W. B. Saunders Company, 1938, p. 705.

4. Eliason, E. L.: Pathological Fractures, Surg., Gynec. & Obst. **56**:504-521 (Feb.) 1933.

5. In a personal communication, Dr. Speed has confirmed this impression.

6. Bowman, F. H.: Pathologic Fracture, U. S. Nav. M. Bull. **35**:73-79 (Jan.) 1937.

again he did not make it clear whether syphilis was acting in the role of a bone-destroying force or whether the fracture resulted from the neurotrophic changes of neurosyphilis. Allen [7] cited Eisendrath's classification of pathologic fractures, in which syphilis was considered among the neuropathies that may give rise to pathologic fracture.

During the past two decades the role of syphilis in the incidence of spontaneous fracture has shown a marked decline. No actual figures are available, but Stokes [8] did not even discuss this phase of syphilis, and Ghormley, Sutherland and Pollock,[9] in reviewing some 660 pathologic fractures in the Mayo Clinic from Jan. 1, 1924, to Jan. 1, 1937, failed to find 1 case of fracture attributable to syphilis of the bone. In the Cincinnati General Hospital approximately 900 fractures are seen each year. In over thirty years' experience in this institution, Caldwell [10] stated that only in the case reported here have gummatous changes in the bones been responsible for fracture. In the literature of the past twenty years few reports are available, and as far as can be determined, there are none in the American literature. It seems safe to assume that this decreased incidence of spontaneous fracture from syphilis of the bone parallels the decreased incidence of syphilis of the bone in general since the introduction and widespread use of arsphenamine, neoarsphenamine and other effective antisyphilitic remedies.

When one considers that the usual changes in the bone in late syphilis (except in cranial lesions) are proliferative rather than destructive, it is not surprising that pathologic fracture is relatively infrequent. In what Stokes called gummatous osteomyelitis proliferative and destructive processes often coexist, and it is this type of pathologic change which is apt to be the basis of spontaneous fracture. In congenital syphilis osteoclastic processes are more common than in the acquired form. But even here spontaneous fractures are rare. This has been brought out by McLean [11] in his excellent work on congenital osseous syphilis and by Tracy [12] and Stewart.[13]

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7. Allen, B.: Pathological Fractures, Radiology **3**:375-376 (Nov.) 1924.

8. Stokes, J. H.: Modern Clinical Syphilology, ed. 2, Philadelphia, W. B. Saunders Company, 1934.

9. Ghormley, R. K.; Sutherland, C. G., and Pollock, G. A.: Pathologic Fractures, J. A. M. A. **109**:2111-2115 (Dec. 25) 1937.

10. Caldwell, J. A.: Personal communication to the author.

11. McLean, S.: Roentgenographic and Pathologic Aspects of Congenital Osseous Syphilis, Am. J. Dis. Child. **41**:130-152 (Jan.) 1931; Correlation of the Roentgenographic and Pathologic Aspects of Congenital Osseous Syphilis, with Particular Reference to the First Months of Life, ibid. **41**:363-395 (Feb.) 1931; Correlation of the Clinical Picture with the Osseous Lesions of Congenital Syphilis as Shown by X-Rays, ibid. **41**:887-922 (April); 1128-1171 (May) 1931.

12. Tracy, F. E.: Advanced Osseous Syphilis in a Four-Months-Old Infant, Radiology **21**:584-587 (Dec.) 1933.

(Footnotes continued on next page)

Experimental data are meager enough. Charpy and Guerin [14] have shown that the bones of syphilitic patients break under less weight and tension than do the bones of normal persons. They also reported some interesting chemical studies of syphilitic bones wherein the fibula of a young person with tertiary syphilis (but without macroscopic involvement of the bones) showed a decreased yield of calcium fluoride.

The clinical picture of syphilitic spontaneous fracture differs in no way from that of similar fracture from other causes, except for the presence of syphilitic osteomyelitis. The fractures are frequently multiple and are often accompanied with osteitis and periostitis of bones not involved by the fractures. Fragenheim said that fractures of the clavicle and ribs may occur as the result of simple muscular activity. The relative order of frequency given by Fragenheim is:

Femur	15
Clavicle	15
Radius	5
Tibia	4
Ribs	2
Patella	1
Ulna	1
Lower jaw	1

The response to therapy is prompt when proper immobilization and antisyphilitic treatment are given.

REPORT OF CASE

Mrs. B. O., a white woman aged 53, was admitted to the Cincinnati General Hospital on Dec. 5, 1937. She gave a history typical of pathologic fracture, having experienced a snapping sensation in her right arm on the day prior to admission, when she attempted to pick up a 4 pound (2 Kg.) package. There had been no similar previous experience, and the patient had been in good health most of her life. There was a history of recurrent spontaneously healing cutaneous ulcers, dating back about ten years, but the past history was otherwise irrelevant. The patient's husband had had some disease of the blood, for which he had received considerable treatment up to the time of his death. There was no other significant item in the family history and no clinical or serologic evidence of syphilis in the patient's 3 children.

Examination showed a fracture of the middle third of the right radius with characteristic deformity. This was confirmed by fluoroscopic study, which also revealed almost complete resorption of the distal part of the ulna. The skin presented numerous atrophic scars characteristic of healed gummas. There was a small superficial ulcer of the nasal septum on the left. The general physical examination gave negative results. None of the viscera were palpable. The

13. Stewart, D. M.: Roentgenological Manifestations in Bone Syphilis, Am. J. Roentgenol. **40**:215-223 (Aug.) 1938.

14. Charpy and Guerin, cited by Fragenheim.[2]

neurologic examination showed nothing abnormal. The urine was normal according to the routine tests and the test for Bence Jones protein was repeatedly negative. The Kahn reaction of the blood was 3 plus; the spinal fluid was clear and the dynamics normal and there were no cells. The Pandy, the Wassermann and the colloidal gold test were negative. The red blood cell count was 3,900,000, and the hemoglobin concentration was 70 per cent. The calcium content of the blood serum was 9.8 mg. and the phosphorus content 4.2 mg. per hundred cubic centimeters. A smear from the ulceration of the nose did not reveal any type of acid-fast bacillus.

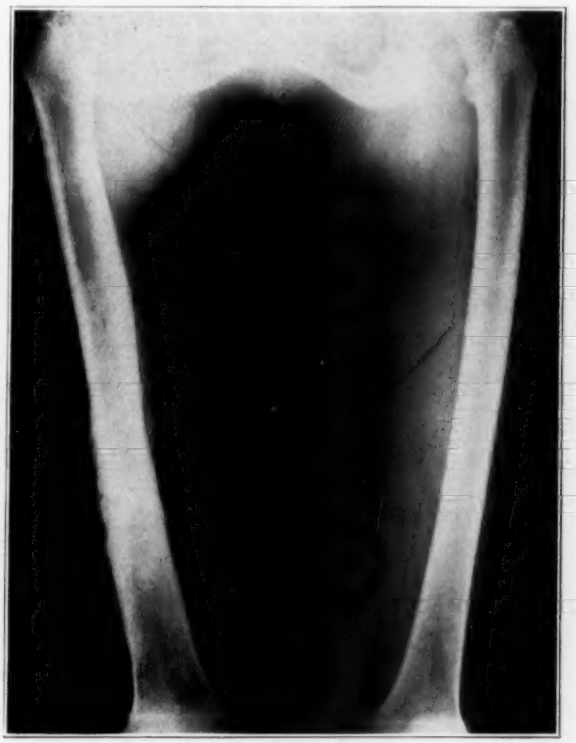

Fig. 1.—Incomplete fracture of the right femur, osteitis and periostitis, in December 1937.

On the patient's admission to the hospital roentgenograms of the right arm confirmed the fluoroscopic findings. Roentgen study of the other bones revealed diffuse osteitis and periostitis of the tibias, humeri and femurs, and an incomplete fracture of the right femur (fig. 1). The skull, pelvis, vertebrae, fingers and toes were uninvolved. In the involved bones there was a preponderance of proliferative rather than destructive reaction.

The diagnosis of multiple skeletal gummas with pathologic fracture of the right radius was made, but in view of the bizarre changes it was deemed necessary to consider other possibilities, such as leprosy and syringomyelia. The nega-

tive nasal smear, the absence of changes in the distal phalanges and of neurologic findings and the negative results of examination of the spinal fluid seemed to exclude these diagnoses satisfactorily.

The patient was put to bed with her arm in a cast, and antisyphilitic treatment was begun. Large doses of potassium iodide were given, and neoarsphenamine and a bismuth preparation were administered. The patient was given eight injections of bismuth subsalicylate and five injections of neoarsphenamine (0.6 Gm. each) without untoward reaction. She was then transferred to the Hamilton County Home and Chronic Disease Hospital, where therapy was continued until bismuth stomatitis developed. On Aug. *26*, 1938, she was readmitted to the Cincinnati General Hospital. Roentgen examination showed almost complete healing of the fractures of the right radius and femur (fig. *2*). There was some decrease in the amount of osteitis and periostitis of the other bones, and the patient

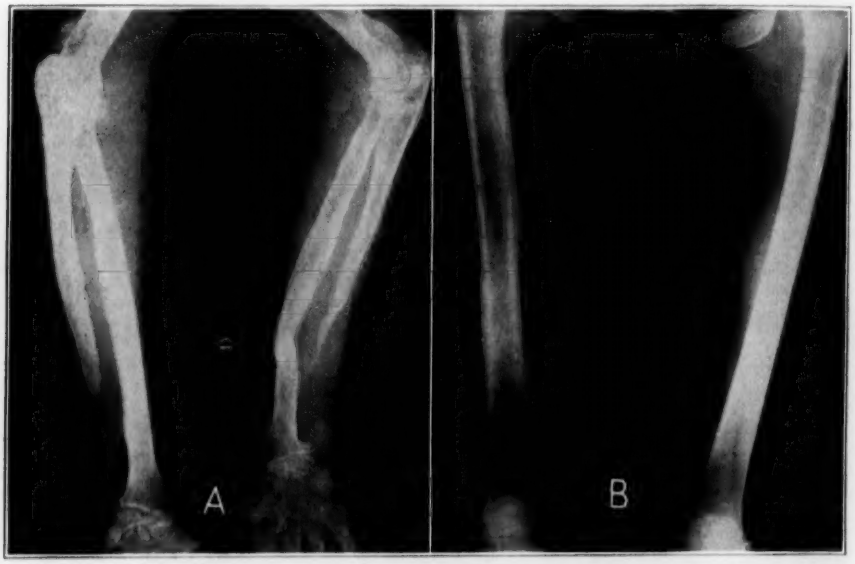

Fig. *2*.—*A*, healing of the fracture and osteitis of the arms, in August 1938; *B*, healing of the fracture and osteoperiostitis of the femur, in June 1938.

was able to walk about the ward. On this second admission the patient complained of persistent "bloating." Examination showed marked hepatomegaly and splenomegaly. The spleen was much larger than the liver and felt somewhat nodular. These findings had not been made on the previous admission. There was no fluid in the abdomen. The red blood cell count was now 3,500,000, with 65 per cent hemoglobin; the white blood cell count was 5,500, with a normal differential count. The urine showed occasional moderate amounts of albumin, and again the test for Bence Jones protein was repeatedly negative. The blood calcium and phosphorus were within normal limits; the spinal fluid was completely normal. Studies of hepatic function (including the congo red test) gave normal results. Roentgenograms of the gastrointestinal tract were normal. Studies of blood and bone marrow were performed by Dr. Harold S. Schiro, of the department of

internal medicine, and interpreted by him as follows: "There is moderate anemia, with a greater deficit of hemoglobin than of red cells. The granulocytes and platelets are normal. The bone marrow shows a slight increase in nucleated red cells which mature normally—a physiologic response to anemia. There is no evidence of leukemia or plasma cell myeloma." It was not possible to ascribe any diagnosis other than syphilis to the visceral lesions, although malignant change, amyloidosis and blood dyscrasia were considered and ruled out on appropriate clinical and laboratory grounds. The anemia responded well to iron therapy, but the hepatomegaly and splenomegaly have remained unchanged during the past six months, despite intensive antisyphilitic treatment. At this writing the patient has had a total of fifteen injections of neoarsphenamine, 0.6 Gm. each, and twenty-one injections of bismuth subsalicylate. At the present time she feels well, performs her daily household duties without difficulty and attends the clinic regularly for treatment and further observation.

COMMENT

This case demonstrates again the frequent association of late skeletal and cutaneous syphilis and the protective role which such lesions apparently exert for the central nervous system. These things have been repeatedly emphasized by Stokes and other syphilologists. The periostitis and osteomyelitis involving the long bones are typical of tertiary syphilis from both the clinical and the roentgenologic standpoint. The prompt response to therapy is also characteristic. The rarefying lesions of the radii and particularly of the ulnas were striking and can be classified as examples of gummatous osteomyelitis. While the final diagnosis for the hepatomegaly and splenomegaly must rest with subsequent events, syphilis is the diagnosis of choice at this time. In view of the absence of evidence of portal obstruction, it was felt that vigorous antisyphilitic (arsphenamine) therapy was not contraindicated. The disappearance of the sensations of "bloating" and fulness in the abdomen, despite the fact that the liver and spleen had not changed objectively, justified this course. The healing of the fractures and of the osteitis and periostitis and the conversion of a bedridden patient to an ambulatory one were truly dramatic.

SUMMARY AND CONCLUSIONS

Pathologic fracture on the basis of skeletal syphilis per se is increasingly less common and is to be differentiated from pathologic fracture as a result of the neurotrophic changes of cerebrospinal syphilis.

The decreased number of cases of pathologic fracture due to syphilis of the bone probably parallels the general decreased incidence of syphilis of the bone of the postarsphenamine era.

A case of gummatous osteomyelitis with pathologic fracture and with interesting visceral complications is presented.

Mr. Joseph P. Homan and Mr. John Bledsoe, of the department of medical art and photography, supplied the photographs used in this report.

LICHENIFICATIO GIGANTEA

(LICHÉNIFICATION GÉANTE OF BROCQ AND PAUTRIER)

CHAIM BERLIN, M.D.
TEL AVIV, PALESTINE

During the past year I had the opportunity of observing simultaneously 2 cases of lichenificatio gigantea, which Brocq and Pautrier [1] and most authors have called a peculiar variety of lichenification. Because of the rarity of this disease, the following 2 cases seem worthy of report:

REPORT OF CASES

CASE 1.—*History.*—R. C., a Jewess aged 82, a native of Russia, had lived 12 years in Palestine. She was first seen in the dermatologic clinic of the Municipal Hospital Hadassah on March 29, 1936. She presented a genital eruption which was diagnosed as genitocrural eczema, which is often seen in this country. The lesion had begun half a year before, with intense itching. Various local applications had not brought any improvement. The patient had never previously had any cutaneous disease. Her family and personal history were unimportant. She had never been seriously ill, except for prolapse of the uterus and hemorrhoids, from which she had suffered for about eighteen years. Routine examinations of the blood, urine and stools gave negative results. The patient received many types of treatment, including ultraviolet irradiation from a quartz lamp and four doses of roentgen radiation, without relief. She discontinued her visits to the clinic for several months and returned on March 25, 1937, complaining that the condition had become aggravated, that new areas had been involved and that the itching had become unbearable, especially at night. She exhibited on the genitalia a typical picture of lichenificatio gigantea, and was admitted to the hospital.

Physical Examination.—On admission, May 5, 1937, the patient was somewhat thin but looked well for her age. The buccal mucosa was normal; the mouth had been edentulous for eighteen years. The size of the heart was within normal limits, and a soft systolic murmur was heard at the apex. The blood pressure was 160 mm. systolic and 80 mm. diastolic. The lungs were normal. The abdomen was tympanitic throughout; there were no palpable masses or areas of tenderness. The glands were not enlarged. The deep reflexes were normal.

Gynecologic Examination (Dr. Asherman).—The uterus had prolapsed. On its right side there was a round hard smooth tumor the size of an egg (myoma?).

From the Department of Dermatology, Municipal Hospital Hadassah.

1. (*a*) Brocq and Pautrier: Ueber einige anormale Formen von Lichenifikation, Arch. f. Dermat. u. Syph. **99**:421, 1910. (*b*) Pautrier, L. M.: Contribution à l'étude de lichénification anormales: La lichénification hypertrophique, ou géante, Ann. de dermat. et syph. **6**:81, 1925; (*c*) Les lichénifications anormales, Acta dermat.-venereol. **8**:313, 1928; (*d*) Lichénification géante, in Darier, J., and others: Nouvelle pratique dermatologique, Paris, Masson & Cie, 1936, vol. 7, p. 535.

Skin.—On both sides of the neck and on the upper part of the chest were areas of pruritus with eczematization. The right genitocrural region showed ordinary eczema. In the left groin (fig. 1), extending medianward to the labium majus and downward to the inner upper aspect of the thigh, a tumor-like lobulated mass, measuring about 10 by 8 cm., rose more or less abruptly from the skin, the height being from 7 to 10 mm. Furrows of varying depth were seen throughout the lesion, suggestive of the gyri of the brain. On the upper

Fig. 1 (case 1).—Tumor-like lesion in the left groin. Note the furrowed surface and the lichenification of the surrounding skin.

portion one deep furrow caused the formation of a lobule, which lay on the normal skin. On the lower portion the furrows were smaller and less distinct and were mostly covered with bloody crusts. The color varied from pinkish to brownish red; in the groin it was grayish white. The consistency was not uniform: the upper lobule was doughy, like a lipoma, the other mass moderately firm and the grayish white areas almost cartilaginous. The lesion moved with the

surrounding skin, which was erythematous and lichenified, especially on the inner aspect of the thigh. The rest of the skin was normal and somewhat pale and did not show evidence of senile degeneration. The finger nails looked glossy, as if they had been polished.

Laboratory Findings.—The hemoglobin concentration of the blood was 60 per cent, and there were 3,600,000 red blood cells per cubic millimeter. The color index was 0.8. The white blood cell count was 9,100, and the differential count was essentially normal. The rate of sedimentation was ninety minutes (Linzenmeyer). Chemical examination of the blood showed, per hundred cubic centimeters; sugar, 101 mg.; uric acid, 3.3 mg.; urea, 39 mg.; cholesterol, 292 mg.; calcium, 8.5 mg., and chlorides as sodium chloride, 660 mg. The Wassermann reaction of the blood was negative. The urine showed a faint trace of albumin, with 3 to 6 white blood cells per high power field.

Histologic Examination (Dr. Karplus).—A deep specimen was removed for biopsy. This contained a small piece of normal skin and a large area of the tumor-like elevated lesion with slightly furrowed surface. In the deeper level of the normal skin there were small nodules which seemed to be prolongations of the large lesion. The most striking feature of the specimen was the pathologic and "giant" enlargement of the papillary body, which presented a dense cellular infiltrate and was sharply separated from the deeper portions of the corium. The overlying epidermis, intact throughout, was in part hyperkeratotic, especially in the furrows. Acanthosis was present in the rete pegs, which were increased in breadth and in length and in some places lacked a basal cell layer (fig. 2). The junction of the cutis and epidermis was in places sharply marked; elsewhere it was indefinite, owing to the separation of the epithelial elements and to the invasion of the rete cells by the cellular infiltrate from the papillary layer. Leukocytes were also seen in the epidermis. The small nodules likewise showed acanthosis and a moderate infiltration of the papillary body, and this infiltrate was of a chronic inflammatory character, composed largely of round cells and a scattering of leukocytes, of which many were eosinophils; the connective tissue cells were also increased in number. In contrast to the upper layers of the corium, the deeper layer and the subcutis showed only a patchy round cell infiltration around the blood vessels and the sweat gland ducts.

Course.—The patient was discharged from the hospital on May 25, 1937, but she continued to visit the clinic and remained under observation for over a year. The eczematous areas, under the influence of antipruritic lotions and ointments, were entirely healed at the end of one month. The giant lesions in the left groin became progressively flatter, so that after three months they presented the picture of common eczema. When examined in January 1938 the skin in this area was flat and smooth but was vitiligo-like in color.

CASE 2.—*History.*—L. A., a Jew 60 years of age, born in Poland, had lived thirteen years in Palestine. His family and personal history were irrelevant. He had not suffered from cutaneous or venereal diseases. Except that hemorrhoids and prolapse of the anus had troubled him for the past twenty years, his general health had been good. There was no history of ingestion of drugs. About three months before he was first seen, in March 1937, erythematous patches appeared in the genitocrural regions, gradually increasing in size and height. The eruption was accompanied with little, if any, sensation. Because it assumed immense dimensions, he decided to visit the clinic.

Physical Examination.—The man was rather slender, but fairly well nourished. He was not nervous. Many teeth were absent, and several were dirty and carious. The heart was not enlarged; no murmurs were heard, and the rate and rhythm were normal. The blood pressure was 110 systolic and 70 diastolic. The lungs were normal. There was no tenderness of the abdomen, and the liver and spleen showed no abnormality. The glands were not enlarged. The reflexes were normal.

Skin.—Symmetrically located, one in each groin, were two tumor-like flat masses measuring about 10 to 12 cm. in diameter. These extended from the proxi-

Fig. 2 (case 1).—Section under high power, showing round cell infiltrate in the upper layers of the corium, wide and deep rete pegs and lack of basal cell layer in some places. Leukocytes have invaded the epidermis; a furrow with hyperkeratosis is present.

mal inner parts of the thighs to the scrotum, involving the areas typical for tinea cruris (fig. 3). The lesions rose more or less abruptly from the skin, the height being from 5 to 8 mm. Many furrows traversed the surface of the growth, the deepest ones having an almost parallel direction. The color varied from dark brown to dirty gray. They were fairly firm, like verruca vulgaris, and in general appeared to be a coalescence of numerous large warts. The surface was

rough, like a nutmeg grater, and in general its vegetating appearance closely resembled that of a cauliflower. The masses were not tender and moved with the surrounding skin, which was studded with many pinhead-sized nodules, the morphologic picture being that of lichen simplex chronicus. Several nodules near the growth were the size of a pea. Hemorrhoids, prolapsus ani and perianal eczema with hyperpigmentation were present. In addition, scattered over the chest and trunk were the minute spinous follicular nearly skin-colored efflorescences of lichen spinulosus.

Laboratory Findings.—The examination of the blood showed 70 per cent hemoglobin, 3,650,000 erythrocytes and 4,700 leukocytes, with 52 per cent polymorphonuclear neutrophils, 31 per cent lymphocytes, 13 per cent monocytes and 4 per cent eosinophils. The rate of sedimentation was about two hours. Chemical examination of the blood showed, per hundred cubic centimeters: sugar, 84 mg.;

Fig. 3 (case 2).—Verrucous lesions in a typical location, with ordinary lichenification in the vicinity.

uric acid, 3.8 mg.; urea, 43 mg.; cholesterol, 304 mg.; calcium, 9.7 mg.; chlorides as sodium chloride, 630 mg., and diastase, 66 per cent. The Wassermann reaction of the blood was negative. Examinations of urine and stools for ova and parasites gave negative results. Repeated examinations of scrapings of skin revealed no fungi. An intradermal test with 0.1 cc. of a 1:50 dilution of trichophytin produced a negative reaction and a test with a 1:30 dilution a faintly positive delayed reaction.

Histologic Examination (Dr. Karplus).—A large specimen was taken from the edge of the mass; this included several small nodules. The most outstanding changes were located in the papillary body, which was enormously enlarged, owing to a cellular infiltrate (fig. 4). The overlying epidermis was slightly hyperkeratotic in some places, corresponding to the macroscopically visible minute furrows. There were both narrow and wide processes, which extended more or less deeply into the papillary layer. The epidermis in these places was mostly acanthotic. Between these deep processes there were also slender and short ones,

which could hardly be distinguished from those of the normal epidermis. The basal cell layer was only partly well developed, and in many places the cellular infiltrate of the papillary body penetrated the epidermis. The infiltrate consisted mainly of round cells and lay in highly vascular connective tissue. Scattered throughout were leukocytes, eosinophils and numerous pigment cells. This solid infiltrate was sharply separated from that of the lower zone of the corium, which was only patchy, occurring about blood vessels or in the vicinity of the sudoriferous ducts. All these changes ceased on the edge of the tumor-like plaque, but in the areas where small papules were macroscopically seen, the papillary body presented an infiltrate of the same cellular characteristics. Wide epithelial pegs were also seen there.

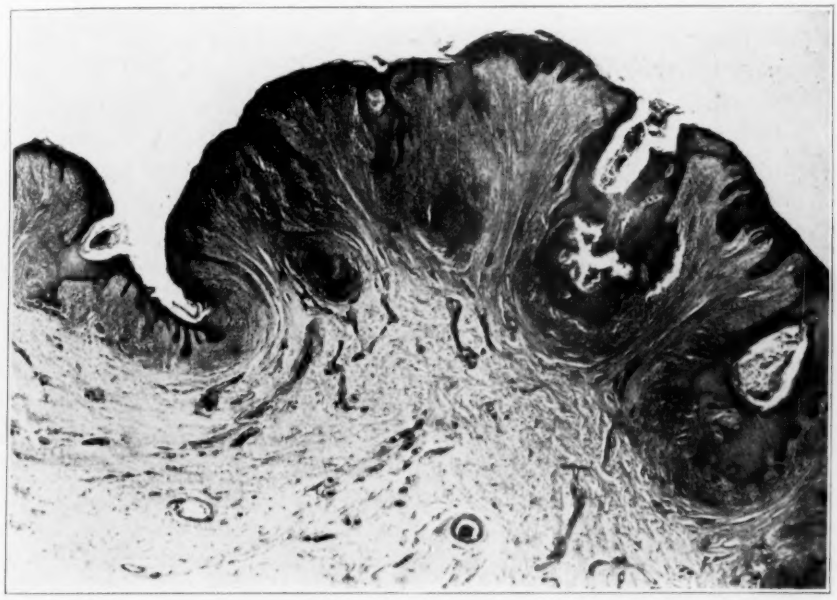

Fig. 4 (case 2).—Section under low power, showing the edge of the tumor-like lesion with a small nodule (left side). There is a dense infiltrate in the papillary body and the upper layer of the corium. Formation of furrows and wide and deep acanthotic rete pegs can be seen.

Treatment and Course.—The patient was treated by a dentist. Many kinds of local medicaments, keratolytic and reducing remedies, such as salicylic acid, tar and chrysarobin, were given without relief. Roentgen therapy (100 roentgens filtered through 1 mm. of aluminum once a week for four weeks) caused improvement. The patient disappeared from observation and was seen again after nine months. He stated that the lesions had undergone resolution several months previously. The areas formerly involved were flat and smooth but decidedly hyperpigmented. In both genital folds the skin was red and moist over an extent of several centimeters and showed a slight degree of intertrigo.

COMMENT

The picture in the first case fitted into the original clinical description of this disorder by Pautrier [1c] in 1928 and was characteristic in all respects. Typical were the intense pruritus which preceded and accompanied the eruption, the manner of development and the localization, size and shape of the tumor-like lesion, which was divided into parts resembling gyri of the brain. In full accord was the histologic picture: acanthosis especially of the rete pegs and the immense infiltration of the upper part of the corium. An unusual feature was the woman's age, 82 years, for the oldest patient until now mentioned in the literature was 70. In case 2, conjointly with some typical points, such as the site of the plaques on the proximal inner parts of the thighs and the scrotum, the size and height of the verrucous and vegetative lesions and the characteristic histologic picture, were some peculiarities, namely, the relative rapidity of development and the almost complete absence of pruritus before and during the eruption. In both my cases there were hemorrhoids and prolapsus ani and in the woman also prolapsus uteri. The third patient of Merenlender,[2] with the site of the lesion on the left labium majus, had also suffered from hemorrhoids and prolapsus ani, and in Werther's [3] case there was a history of varicose veins.

It will be appropriate to note the good prognosis and the complete healing in both my cases, for lichenificatio gigantea is considered to be a chronic disorder which responds slowly to therapy. In several cases it had lasted more than ten years (Merenlender,[2] Sorlat and Drouet [4] and Zoon [5]). There was an impression that in my cases the deep incision for biopsy contributed to the resolution of the lesions.

In 1930 Merenlender listed all the cases of this disorder which were reported in the European literature. The eruption was described under various names, among them papillomatosis cutis, neurodermitis verrucosa, *sarkoide Hauttumoren, pseudoelephantiasis scroti et lichénification,* neurodermia chronica vegetans and neurodermitis gigantea. Merenlender's list contained 26 cases, including his own 3. Since then the following authors have reported this disease: Zoon [5] (1929), Beron [6]

2. Merenlender, J.: Ueber Neurodermitis gigantea (Lichénification géante Pautrier-Brocq), Arch. f. Dermat. u. Syph. **161**:6, 1930.

3. Werther: Fall von Neurodermia chronica vegetans, Zentralbl. f. Haut- u. Geschlechtskr. **30**:434, 1929.

4. Sorlat and Drouet: Lichénification géante et lichen plan buccale, Bull. Soc. franç. de dermat. et syph. **37**:4, 1930.

5. Zoon, J. J.: Lichénification géante, Acta dermat.-venereol. **9**:490, 1929.

6. Beron, B.: Zweiter Beitrag zur Klinik der anormalen Lichenifikation, Dermat. Wchnschr. **90**:233, 1930.

and Sorlat and Drouet [4] (1930), Brünauer [7] (1931), Merenlender [8] (1932), Lutz [9] and Beron [10] (1935), Merenlender [11] (1937) and Engelhardt [12] (1938). In all, I have been able to find 10 reported cases; most of them were presented at various dermatologic meetings. With my additional two cases the total now reported is 38.

With regard to age and localization, the cases of Brocq [13] and Wecker [14] presented an exception. Both patients were children, 1 and 1½ years of age, respectively, and the involvement was generalized. In general, older patients predominated. The age varied from 25 to 82, the average being 51. Especially outstanding were Vollmer's [15] and Fantl's [16] patients, with immense papillomatous changes, particularly on the scrotum but also on other parts of the body and in the former, curiously, on the lips and even in the mouth. In one of Pautrier's [1b] cases, the axillas as well as the genitalia were affected. In Engelhardt's case the lesions were located on the scrotum and on the right wrist, and in the case of Bizzozero and Narducci [17] only the backs of the hands were the seat of the changes. In the other 31 cases only the lower part of the body was involved, chiefly the genitocrural region, which was affected either alone or in association with one other area of the lower extremity. The number of lesions varied from one to several, but frequently only one was present.

Pautrier expressed the opinion that the site of predilection on the genitalia is due to the greater delicateness of the skin in this region. I, however, believe that friction, chafing and irritation from perspiration, sebum and epithelial débris are also responsible, and in this region, therefore, other diseases assume vegetating characteristics. One must think

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7. Brünauer: Lichinificatio gigantea Brocq-Pautrier; Neurodermitis hypertrophica Ehrmann, Zentralbl. f. Haut- u. Geschlechtskr. **35**:343, 1931.

8. Merenlender, J.: Neurodermitis gigantea, Zentralbl. f. Haut- u. Geschlechtskr. **39**:145, 1932.

9. Lutz: Lichénification géante, Zentralbl. f. Haut- u. Geschlechtskr. **49**:122, 1935.

10. Beron, B.: Zwei neue Fälle von anormaler Lichénifikation, Zentralbl. f. Haut- u. Geschlechtskr. **51**:342, 1935.

11. Merenlender, J.: Neurodermitis gigantea, Zentralbl. f. Haut- u. Geschlechtskr. **54**:561, 1937.

12. Engelhardt, W.: Neurodermitis verrucosa seu papillomatosa, Dermat. Wchnschr. **105**:1283, 1937; **106**:304, 1938.

13. Brocq, cited by Merenlender.[2]

14. Wecker, cited by Pautrier.[1d]

15. Vollmer, E.: Ueber Papillomatose, Arch. f. Dermat. u. Syph. **79**:293, 1906.

16. Fantl, G.: Ueber Papillomatosis cutis, Arch. f. Dermat. u. Syph. **129**: 332, 1921.

17. Bizzozero, E., and Narducci, F.: Contribution à l'étude des formes anormales de lichénification, Ann. de dermat. et syph. **9**:857, 1928.

of pemphigus vegetans in the genital region, and condyloma latum and acuminatum, the morphologic picture of which often resembles the disorder under discussion. In the history of the cases of Vollmer, Fantl, Rusch [18] and Kreibich [19] maceration and sweating preceded the giant changes. Striking is the frequent involvement of the scrotum and the corresponding proximal inner aspects of the thighs, present in 19 of 38 cases. This can undoubtedly be explained by the constant peculiar perspiration that occurs in this region. This is also the reason why the disease is more often encountered in men. Of the 38 patients, 28 were men, 7 women, 2 children and 1 of unknown sex.

As to the designation, it is difficult to find a name for this disease which will satisfy all the authors. Before Brocq and Pautrier it was known in the literature under various titles. Zoon [5] recently proposed a new name, prurigo circumscripta papillomatosis sive vegetans, yet it seems to me that the name *lichénification géante* (lichenificatio gigantea) of the French authors is fortunately chosen. The term lichenificatio indicates the origin of the disease, from pruritus and scratching, and the surrounding skin frequently looks typically lichenified; the word gigantea signifies the monstrousness of these changes.

SUMMARY AND CONCLUSIONS

Two cases of lichenificatio gigantea are described in detail. One patient was a woman 82 years of age, and the other was a man of 60.

The good prognosis in both cases is emphasized.

It is pointed out that sweat contributes to the formation of the giant changes. The preference of the disorder for the genital region, especially the male groin, is, therefore, explained.

Merenlender's collection of 26 cases up to 1930 is now enlarged by 10 cases subsequently reported in the literature and 2 reported here, making a total of 38 cases.

The name lichenificatio gigantea is fortunately chosen and should be accepted.

9 Montefiore Street.

18. Rusch, P.: Zur Kenntnis der "sarkoiden" Hauttumoren, Arch. f. Dermat. u. Syph. **87**:163, 1907.

19. Kreibich, C.: Neurodermitis verrucosa, Arch. f. Dermat. u. Syph. **121**: 307, 1916.

SCLERODERMA ASSOCIATED WITH ADRENAL NEOPLASM

REPORT OF A CASE

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ENGLEWOOD, N. J.

Statements are made in textbooks of medicine and in the literature concerning the possible relation between scleroderma and pathologic changes of the adrenal glands. This point has never been thoroughly settled, and the cause of scleroderma remains a mystery.

In searching the literature of the past fifteen years I found only two reports specifically mentioning a relation of this disease to pathologic changes of the adrenal glands, and these were in the foreign literature.[1]

For this reason, the following case is presented with the necropsy report.

REPORT OF CASE

A. K., a German-born widow aged 59, had lived in the United States for thirty-seven years. She came to the Englewood Hospital on Dec. 1, 1936, with chief complaints of pain in the chest for two weeks, dyspnea on exertion for one year or more and stiffness of the joints with discoloration of the skin, which had been progressive during the past two years.

She stated that she had first noticed poor health about two years previously, when articular symptoms developed, first appearing as stiffness of both hands. Coexistent with the development of these symptoms there had been a progressive discoloration of the skin. The symptoms in the joints progressed up to the time of admission, in spite of treatment with injections (preparation used not known) by two local physicians, oral medication prescribed by a third physician and a course of chiropractic treatment. Some time after the symptoms became apparent she noticed increasing dyspnea on exertion, which became progressively worse, until she was unable to walk any appreciable distance. (She could not state whether this was the result of dyspnea or stiffness of the joints or both.) Two weeks before admission she was seized with a constricting precordial pain, which caused her to

From the Department of Medicine, Englewood Hospital.

1. Scholz, K.: Combination of Scleroderma with Addison's Disease; Combination of Scleroderma with Raynaud's Disease and Tendovaginitis Crepitans, Klin. Wchnschr. **1**:1948 (Sept. 23) 1922. Devoto, A.: Case of Scleroderma Affecting the Finger: Improvement Under Polyglandular Treatment, Especially Adrenal Treatment, Gior. ital. di dermat. e sif. **66**:1071 (June) 1925.

stand still, unable to move. The pain did not radiate. She reported two similar, milder attacks of sudden precordial pain during the previous year.

The patient stated that she had enjoyed good health until two years before. Since then she had had innumerable worries. Her husband had died about one and one-half years previously, and she felt that this shock definitely increased the development of her symptoms. During the preceding three months all of her general symptoms (aside from the acute pain in the chest) had become worse, until she was practically bedridden.

After the symptoms appeared in her hands, a tenseness of the skin on the abdomen and hips became apparent to her. There had been definite amelioration of symptoms in warm weather, with an increase in cold weather. Immersion of the hands in cold water rendered them cyanotic at any time during the entire period of her illness. An apparent thickening of the tongue was also noted, which at times produced difficulty in speech.

At the time of the onset of pain in the chest she had a moderately severe attack of diarrhea (type unknown.)

The family history was irrelevant.

Past and Personal History.—An operation for femoral hernia had been performed twenty-two years before, and she had had two uneventful pregnancies. Menstruation had been regular until the menopause, which had appeared ten years before, without undue trouble. She did not smoke and took no alcohol or drugs except those prescribed by physicians. She had eaten rye bread all her life.

Physical Examination.—The patient was bedridden and suffered from slight orthopnea. The skin presented a dark brownish discoloration most pronounced on the neck, the upper part of the chest, the forearms, the abdomen, the thighs and the legs. The skin in these regions, particularly on the forearms, lower part of the abdomen, left thigh and legs, was tense, smooth and glistening and appeared thin. The face had a waxy appearance, with what seemed to be an early stage of risus sardonicus. The region around the mouth appeared pinched and drawn; the eyes reacted normally, and the pupils were equal; the extraocular movements were normal; the teeth were in poor condition; the mucous membranes were normal. The thyroid was hard, enlarged and smooth on the left side. There was no adenopathy or venous distention. The sternum was prominent, the ribs presenting a contour resembling healed rickets; a diffuse apical beat was noted. There were increased voice sounds in the right axilla, with a slight bronchovesicular quality of the breath sounds. The heart was enlarged to the left and downward; the blood pressure was 118 systolic and 84 diastolic, and the rhythm was irregular, with an occasional forceful beat at the apex; no murmurs were noted, and the sounds were distant. The liver was 3 or 4 cm. below the costal margin and was smooth and hard; the arms were fixed in moderate flexion at the elbow, and the fingers were flexed. The knuckles of both hands were swollen, and there were small ulcerations on the right index and the left middle finger; the arms were fixed at the shoulders, the muscles of the arms and forearms appearing fibrous to the touch; the legs were fixed in flexion at the knee, and the knee joints were swollen and nontender; the ankles and feet were swollen, and the toes were cyanotic; there was no pulsation of the dorsalis pedis or the posterior tibial artery of either side (but the skin was tense on the dorsa of the feet). The muscles of the lower extremities had the same feel as those of the upper, but to a lesser extent. The fingers were typically sclerodactylic, with a tense marble-

like feel to the terminal pulps, extending back to the proximal metacarpal-phalangeal region; the palms had a similar tenseness; the pulps of the fingers were whitish and bore no circulatory color changes whatever.

The clinical diagnoses were (1) generalized arteriosclerosis, (2) arteriosclerotic heart disease, (3) possible coronary occlusion, (4) scleroderma with sclerodactylia and (5) adrenal insufficiency(?).

Roentgen Examinations.—Pictures of the skull, extremities and abdomen revealed no abnormalities except marked arthritic changes in the extremities.

Laboratory Findings.—Chemical study of the blood, the blood count, urinalyses and determinations of basal metabolism all gave results within normal limits. There was mild leukocytosis. An electrocardiogram showed myocardial disease and left bundle branch block.

Course in the Hospital.—The patient was hospitalized for twenty-four days, without any particular developments, and died suddenly on the twenty-fourth day, presumably from cardiac failure. Her only subjective complaint during hospitalization was of stiffness in the affected joints. While she was in the hospital, her waxlike facial appearance seemed to become more pronounced. Death occurred on Jan. 4, 1937, before any attempt was made toward therapy for the scleroderma, and she had purposely been kept quiet because of her cardiac condition.

Autopsy Report (by Francis J. Fadden, M.D., pathologist).—Anatomic diagnoses were adenoma of the left adrenal gland, chronic myocardial degeneration, splenitis, adenomatous goiter, cysts of both kidneys, pleuritis, hydrothorax and arteriosclerosis.

The body was that of a well developed, poorly nourished white woman, with markedly bronzed skin with a glistening sheen. The skin was firm and thickened; the arms and fingers were flexed. The knuckles and elbows were swollen, as were the knees. There was callus formation over the finger tips and the knuckles, and the legs showed old scars of ulcers. The face was masklike, the hair dry, brittle and gray and the skin bronzed. The right eye showed corneal opacity, but the pupils were equal. The nose was normal, the teeth poor and the lips dry.

The primary incision was of Y type, and the panniculus adiposus was 0.5 cm. thick. The muscles were firm and adherent to the surrounding tissues.

The peritoneal cavity was smooth and glistening. There were no adhesions and no free fluid. The appendix was present and normal.

The right pleural cavity contained 200 cc. of thin pale fluid and old fibrous adhesions posteriorly at the base. The left pleural cavity was similar to the right, except that there were no adhesions.

The pericardial cavity was smooth and glistening, with a normal amount of straw-colored fluid.

The heart weighed 420 Gm.; the epicardium was dark red, and there were irregular gray plaques 2 cm. in diameter over the entire area. The coronary arteries were beaded and firm; they were sclerotic but patent. The measurements of the circumference of the valves were: tricuspid valve 10 cm., pulmonic valve 7 cm., mitral valve 12 cm., and aortic valve 9 cm. The wall of the right ventricle was 0.4 cm. thick, and that of the left ventricle, 2 cm. The myocardium showed areas of gray fibrosis, and the ventricles were thin, with soft musculature. The endocardium was dark red and smooth. The mitral valve was thickened and the aortic valve sclerotic.

The right lung was spongy, gray and smooth. Sections of it showed gray, crepitant and subpleural anthracotic markings. A frothy fluid could be squeezed from the bronchioles. The left lung was similar to the right.

The spleen weighed 320 Gm. and was firm and dark red. Sections of it showed a fairly firm, dark red pulp. Scant pulp scraped away on the knife.

The pancreas weighed 90 Gm. and was firm, yellow and lobulated. Its sections showed a yellow lobulated surface.

The gastrointestinal tract presented no adhesions, no obstructions and no distention.

The liver weighed 1,500 Gm. and was dark red, with a smooth capsule. Sections were greasy.

The gallbladder was filled with dark green bile. There were no stones, and the ducts were patent.

The left adrenal gland, showing cut section of the adenoma.

The right adrenal gland weighed 11 Gm. and was firm and yellow. Its sections showed normal medullary and cortical portions. The left one weighed 20 Gm., measured 7 by 3.5 cm. and was firm and yellow. Its sections showed a yellow encapsulated friable nodule 3.5 cm. in diameter in the medullary portion.

The right kidney weighed 290 Gm. and was firm and dark red. Its sections showed its capsule to strip easily from a smooth surface. There was good medullary and cortical differentiation. There were several cysts 2 to 6 cm. in diameter, which were smooth walled and filled with pale fluid. The left kidney weighed 285 Gm. and was similar to the right.

The ureters and the bladder were normal. The genitalia were atrophied. The veins and the lymph nodes were normal. The thyroid weighed 65 Gm. and was dark red, with fluctuant areas. Sections of it showed the parenchyma to be replaced with cysts filled with thin, pale fluid.

The aorta was sclerotic.

Death was due to chronic myocardial degeneration with failure.

HEREDITARY MULTIPLE TELANGIECTASIA

(RENDU-OSLER DISEASE)

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Multiple telangiectasia occurring in families and passing from generation to generation through heredity has been observed for many years and reported under different names. Originally the disease was considered to be a form of hemophilia, because of the frequent hemorrhages the patients suffer, but in 1896 Rendu [1] first separated it from hemophilia, reporting a case under the name "Epistaxis répétées chez un sujet porteur de petits angiomes cutanés et muqueux." It remained for Osler [2] to create the clinical entity known at present, when in 1901 he published his first report, under the title "On a Family Form of Recurring Epistaxis with Multiple Telangiectases of the Skin and Mucous Membranes."

Since the time of Osler's publication the disease has been observed from time to time and his conclusions amply confirmed. A complete review of the literature may be found in Goldstein's articles.[3] According to Goldstein, perhaps 110 authenticated and accepted instances have been recorded in the entire medical literature of the world, with mention of about 650 affected persons.

CLINICAL DESCRIPTION

The disease is congenital and appears in children and in several members of a family. Often the first manifestations consist of hemorrhages, apparently without any cause, and usually epistaxis but also

1. Rendu: Epistaxis répétées chez un sujet porteur de petits angiomes cutanés et muqueux, Bull. et mém. Soc. méd. d. hôp. de Paris **13**:731 (Oct. 23) 1896.

2. Osler, W.: On a Family Form of Recurring Epistaxis with Multiple Telangiectasis of the Skin and Mucous Membranes, Bull. Johns Hopkins Hosp. **12**: 333 (Nov.) 1901.

3. Goldstein, H. I.: Hereditary Multiple Telangiectasia: Goldstein's Heredofamilial Angiomatosis with Familial Hemorrhages or Rendu-Osler-Weber's Disease, Arch. Dermat. & Syph. **26**:282 (Aug.) 1932; Goldstein's Disease or Rendu-Osler-Weber's Disease, Acta dermat.-venereol. **13**:661, 1932; in Deliberationes Congressus dermatologorum internationalis, Leipzig, Johann Ambrosius Barth, 1935, vol. 1, p. 756.

bleeding of the gums, pharynx, rectum, stomach or other mucous membranes. In some cases the small, repeated hemorrhages are the only symptoms for several years, the cutaneous phenomena appearing later in life. In other cases the cutaneous and mucosal manifestations are predominant and consist of telangiectases, stellar angiomas and small ruby points. These lesions are situated on the face, chest, arms, mucous membranes of the lips and septum of the nose and in some cases elsewhere on the skin or mucous surfaces. The size of the lesions varies from that of fine threads or small reddish puncta to 4 or 5 mm. in diameter; mostly they are dark red threadlike telangiectases, which sometimes coalesce to form spider-like designs, netlike nevi and brilliant red pinpoint-sized angiomas. In rare cases large port wine nevi occupy large surfaces of the skin. On the lips the lesions are punctate and slightly prominent. These punctiform nevi are sometimes pulsating when observed through a diascope, but we have not observed this in any of our cases. The tongue, gums, palate, pharynx and conjunctivas may present delicate vascular dilatations, and the mucosa of the nose, especially on the septum, may present telangiectases which burst easily, with subsequent bleeding. Varices of the legs and hemorrhoids have been reported in some cases. Bleeding from the rectum attributed to hemorrhoids may be really caused by the bursting of small telangiectases of the mucosa of the anus.

Most patients suffer repeatedly from more or less abundant and violent hemorrhages leading to secondary anemia, and sometimes mortal hemoptysis, hematemesis and metrorrhagia have been reported. However, many patients have typical familial, hereditary cutaneous and mucosal lesions which never bleed. Such cases have been reported by Ballantyne,[4] Laffont,[4] East,[4] Madden [5] and others. Two of the cases to be reported later belong to this category. On the other hand, many cases have been reported in which some members of a family had hemorrhages as the main feature and no telangiectases, while other members of the same family presented cutaneous lesions as well as hemorrhages.

Purpuric lesions have never been observed; the coagulation time and bleeding time are always normal; the blood count presents no changes, and the thrombocyte level is within normal limits. There is no vascular fragility, as demonstrated by a negative reaction to the arm band test. The liver and spleen are usually unaffected, but some

4. Cited by Perin, in Darier, J., and others: Nouvelle pratique dermatologique, Paris, Masson & Cie, 1936, vol. 6, p. 509.

5. Madden, J. F., in discussion on Lawless, T. K.: Telangiectasia (Hereditary, Hemorrhagic)? Osler's "Recurring Epistaxis with Multiple Telangiectasis of the Skin and Mucous Membranes"? Arch. Dermat. & Syph. **30**:295 (Aug.) 1934.

cases have been reported in which there was enlargement of these organs, probably owing to the long existing and repeated hemorrhages. Secondary anemia may be severe in cases of chronic bleeding.

ETIOLOGY

The disease has been definitely separated from hemophilia, purpura haemorrhagica and other blood dyscrasias. It seems to be accepted by the majority of observers that the condition is due to a congenital defect of development of the terminal loops of the capillaries. This defect is transmitted by heredity and considered a dominant characteristic, not sex linked and therefore different from other diseases of the blood vessels or the blood with bleeding tendencies (Ullmann [6]). Both sexes may be affected, but the female is affected more frequently. The number of persons affected in a family is approximately one third, and the condition may be traced through three or four generations. Syphilis, tuberculosis, alcoholism and hepatic disease seem to play no part in the causation. There are no disturbances of menstruation, and childbirth occurs without complications. According to Aubertin and his associates,[7] the condition may be aggravated after the menopause.

The condition progresses throughout life with no tendency to spontaneous involution; it may be benign and the patient hardly aware of its existence, but it may have a fatal ending due to hemorrhage or secondary anemia.

PATHOLOGY

Most writers have reported vascular dilatations in the form of lakes in the papillary and subpapillary layers. The vessel walls have only a row of endothelial cells and are limited by a thin connective tissue structure without elastic fibers or muscular coat. The epithelium is thinned but otherwise normal. According to Ullmann,[6] the condition is a disease of the blood vessels and not of the blood, the main feature being the lack of elastic tissue in the walls of the capillaries and in the arterioles and venules, which causes an increased vulnerability or fragility of the blood vessels with a tendency to hemorrhages.

According to Neumark [8] histologic examination of his sections showed, in addition to dilatation and hyperplasia of the blood vessels,

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6. Ullmann: Angiomatosis (Morbus Osler): Ist sie eine Blut- oder Gefässerkrankung? in Deliberationes Congressus dermatologorum internationalis, Leipzig, Johann Ambrosius Barth, 1936, vol. 2, p. 743.

7. Aubertin, C.; Lévy, R., and Baclesse: L'angiomatose hémorragique familiale (maladie de Rendu-Osler), Presse méd. **41**:185 (Feb. 4) 1933.

8. Neumark: La maladie d'Osler, in Deliberationes Congressus dermatologorum internationalis, Leipzig, Johann Ambrosius Barth, 1936, vol. 2, p. 746.

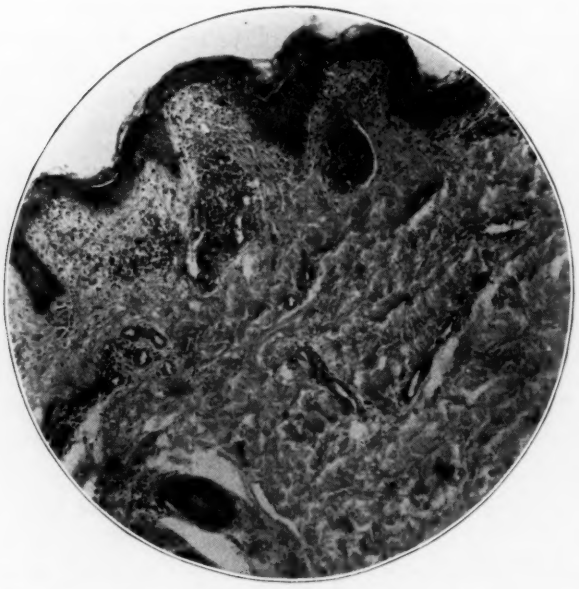

Fig. 1.—Photomicrograph showing dilated capillaries in the upper part of the cutis, surrounded by proliferation of adventitial cells.

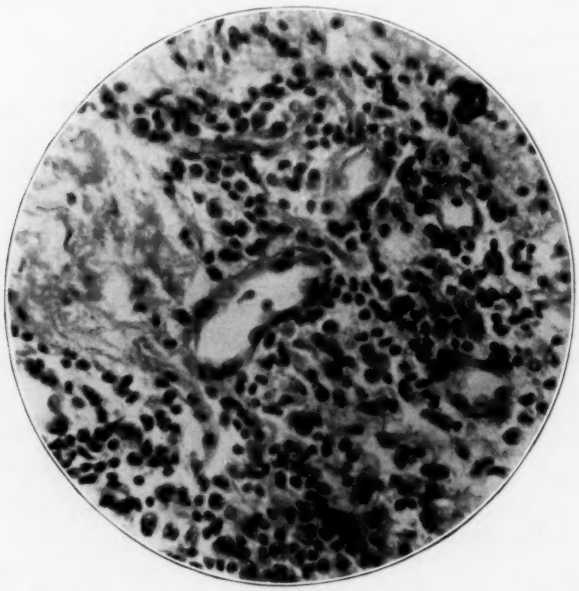

Fig. 2.—Photomicrograph showing dilated capillaries and the character of the proliferating cells.

distinct changes in the connective tissue. These changes may be considered in two groups: the first includes those of degenerative nature, consisting of edema and disintegration of the conjunctive fibers and elastorrhexis and even absence of the elastic fibers. These changes explain the passive dilatation of the veins and of the capillaries (telan-

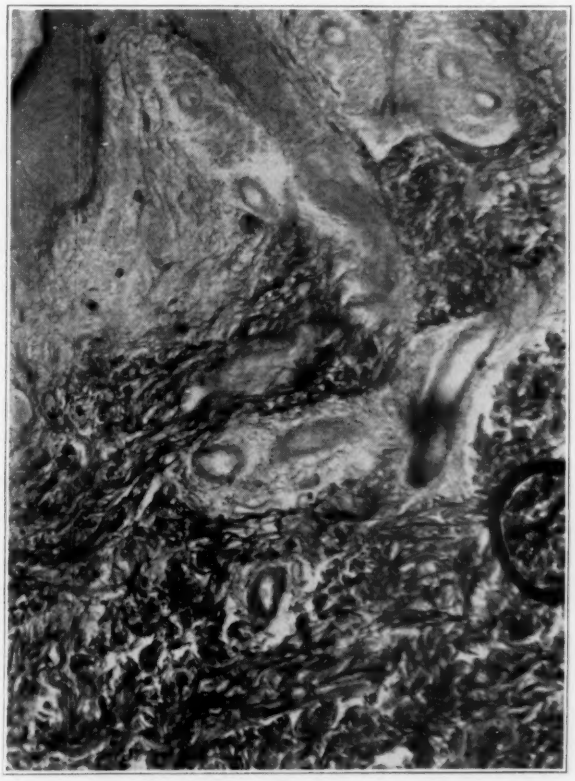

Fig. 3.—Photomicrograph showing absence of elastic tissue in and around blood vessels; Weigert stain.

giectases) and the tendency to hemorrhages, usually due to trauma. The second group consists of the presence of numerous newly formed vascular buds; of numerous cellular bands composed of cells with round or oval nuclei (angioblasts), among which may be seen lacunar spaces partially lined with endothelial cells; multiplication of connective tissue cells, and of numerous mast cells.

Most of these features have been confirmed by us, as will be set out later.

REPORT OF CASES

We have had the opportunity to examine three families affected with multiple hereditary telangiectases. Of the members affected we have chosen 1 of each family and endeavored to exhaust the study of his disease. Of family 1, we examined 4 members; of family 2, we examined 2 members; and of family 3, we examined only 1 member.

FAMILY 1.—Four members of one generation were examined. The family consisted of the mother, 3 daughters and 2 sons. The mother, 2 daughters and a son were affected; 1 daughter was normal and 1 son's condition was unknown, since he was not available for examination and the mother could give no definite information (fig. 4).

A daughter aged 12, white, was chosen for study. She had been born in Cuba of Spanish ancestors. According to the mother, the child had presented the vascular spots since babyhood, and her other 3 children presented the same cutaneous anomalies. She herself had observed these vascular dilatations on her skin since early childhood but had never attached any importance to them. She

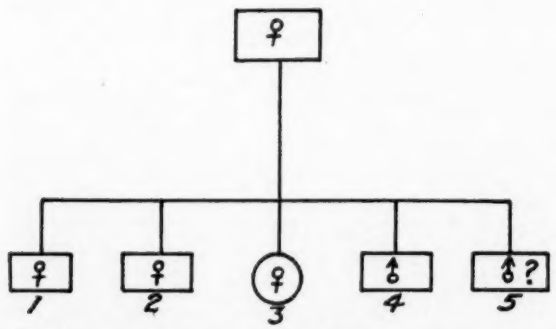

Fig. 4 (family 1).—Genealogic chart. In this and subsequent figures a rectangle indicates an affected person; a circle, a normal one.

was an only daughter and did not remember if her parents had this condition. Her first child, the subject of study, had always been healthy except for diseased adenoids and tonsils, which were removed at the age of 8 years without complications or abnormal bleeding.

Examination.—On the face, particularly on the nose, cheeks and lips, the patient presented numerous telangiectases, red puncta and small stellar and arachnoid nevi. Similar lesions could be observed on the neck, upper part of the thorax, shoulders and arms. On the mucocutaneous juncture of both lips there were rows of punctiform vascular dilatations of ruby color, the size of pinpoints, symmetrically arranged. On both upper and lower limbs vascular points were observed also, in the form of minute points surrounded by anemic halos about 3 to 5 mm. in diameter, which became more visible on exposure to the cold air and when the patient became emotional. Several stellar angiomas were distributed about both arms and legs. The surface of the skin in these locations resembled that of livedo racemosa, owing to the numerous anemic spots around the dilated vessels. On the outer surface of the left elbow there was a port wine nevus 2 cm. wide.

Otorhinolaryngologic examination showed no appreciable telangiectases on the mucous membranes of the nose, mouth, larynx and pharynx. The fundi were

normal, and the conjunctivas showed slight vascular congestion. General examination of abdominal and thoracic organs showed no abnormality. The child was well developed for her age. The blood pressure was 100 systolic and 70 diastolic.

Laboratory Tests: The Kahn and the Meinicke test were negative. A blood count showed 70 per cent hemoglobin, 4,060,000 red cells and 12,000 white cells, with 4 per cent eosinophils, 67 per cent polymorphonuclears, 25 per cent lymphocytes and 4 per cent monocytes. The bleeding time was two minutes (Duke); the coagulation time, one and one-half minutes (Sabrazes), and the platelet count, 274,540. Arm band compression gave negative results. The injection of 1 mg. of epinephrine hydrochloride subcutaneously caused a most curious and unexpected phenomenon. At the end of one hour the telangiectases became turgescent; new vascular spots appeared on the chest and face, and the anemic halos surrounding the lesions on the arms and legs became more marked. The face had the aspect of an acute rash at a distance resembling measles. Two hours later the skin returned to its usual condition. Injections of epinephrine hydrochloride were given every day for twenty days, and the same transitory phenomena were observed every time. There was no definite change of the condition, the effects of the epinephrine hydrochloride disappearing in a few hours.

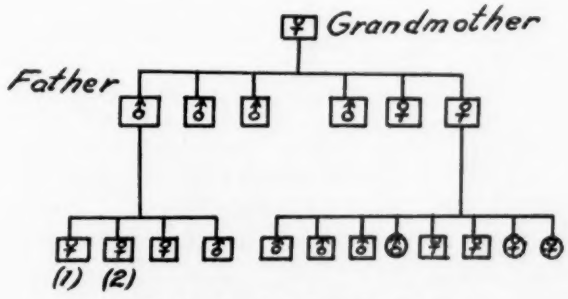

Fig. 5 (family 2).—Genealogic chart.

FAMILY 2.—In this family the telangiectatic manifestations were present in three generations, affecting 15 of a total of 19 members. Of these only 2 with typical Rendu-Osler disease were seen. The patient chosen for study was a white girl aged 18, born in Cuba of Spanish ancestors, a student at normal school (fig. 5). The cutaneous lesions had existed since early childhood, causing no inconvenience although they appeared more prominent on the days preceding menstruation and during emotional outbursts. The patient never suffered from epistaxis or other hemorrhages; her menses were of normal amount and duration and she had always been healthy, having suffered only from measles, whooping cough and chickenpox in childhood. Her father was alive and healthy, was 47 years old and presented the same type of cutaneous telangiectases and nevi, but had never had any hemorrhages. There were 2 more sisters and a brother; only the sisters presented telangiectases and spider nevi, although in small numbers. All the patient's uncles and aunts on the father's side, 3 nephews and 2 nieces also showed telangiectatic lesions but no hemorrhages.

Examination.—On the face and particularly on the forehead, nose and cheeks there were innumerable capillary dilatations in the form of striae and puncta, telangiectatic networks and spider nevi. The puncta were surrounded by light anemic halos. The color of these lesions was bright red, disappearing on pressure

but only momentarily, the vessels filling again as soon as pressure was removed. Around the mouth and on the mucocutaneous juncture there were rows of puncta symmetrically situated on both sides of the median line. On the neck there were a small flat nevus about 1 cm. in diameter and a few telangiectases. On the chest, especially on the upper part, the telangiectases were numerous, gathering in several places to form networks, and spider and stellar nevi. On the arms there were many cayenne pepper spots surrounded by anemic halos. On the rest of the cutaneous surface the vascular lesions were sparse.

Examination of the nose, mouth and throat gave negative results. Ophthalmologic examination also showed no abnormalities. The internal organs were normal. The blood pressure was 125 systolic and 85 diastolic.

Laboratory Tests: The Kahn and the Meinicke test were negative. A blood count showed 75 per cent hemoglobin, 4,430,000 red cells and 11,500 white cells, with 3 per cent eosinophils, 71 per cent polymorphonuclears, 22 per cent lymphocytes and 4 per cent monocytes. The coagulation time was one minute (Sabrazes); the bleeding time, one and one-half minutes (Duke). Arm band compression gave negative results. The injection of 1 mg. of epinephrine hydrochloride produced the same phenomena described in the previous case.

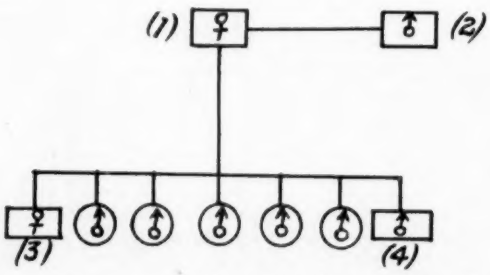

Fig. 6 (family 3).—Genealogic chart.

FAMILY 3.—The condition existed in two generations, but only 1 member of the family could be examined and studied. In 2 of the affected members, a maternal uncle and a brother, only epistaxis was present; the other 2, the patient studied and her mother, had both telangiectases and hemorrhages (fig. 6).

The patient was a white girl aged 19, born in Cuba of Spanish ancestors, single, a house worker. She entered the surgical ward of the Mercedes Hospital with elephantiasis of the left lower extremity, chyluria and a history of rectal, oral and nasal bleeding since childhood. Dr. F. Leza,[9] professor of surgery, performed an operation on the leg, removing a large amount of the hypertrophic skin and subcutaneous tissue, without any complications.

The patient stated that she had had telangiectases, spider nevi and large port wine nevi as long as she could remember. From about the age of 12 she had suffered from slight attacks of epistaxis, which were easily controlled and which had become less frequent in the last five or six years. For the past three or four years she had had rectal hemorrhages every month or two, sometimes twice a month, and occasionally she had seen her saliva tinged with blood and had noticed the taste of blood in her mouth, but not until recently had this amounted to a

9. Dr. Leza permitted us to study this patient from a dermatologic point of view.

hemorrhage. Lately oral bleeding had become an almost daily occurrence, and the patient had become pale and weak.

On examination the skin of the face, thorax and extremities showed numerous telangiectases in the form of small red puncta surrounded by anemic halos, thread-like and spider-like nevi and here and there small ruby-colored angiomas. On the mucocutaneous juncture of the lips there were dilated capillaries, most of them in the form of ruby points. The right hand, forearm and arm were the seat of a large flat nevus of a dull bluish red. Practically all of the right lower extremity presented a livid angiomatous surface. It is to be noted that this leg was the seat of an enormous elephantiasis, already mentioned. On the left lower extremity there was likewise a large nonelevated angiomatous surface of livid color, reaching from the upper third of the thigh to the last four toes and the sole, only small surfaces of the posterior region of the leg being normal.

These lesions varied in color according to the emotional state of the patient and to the temperature of the air, the anemic halos around the punctiform lesions being more marked in cold weather and during excitement, when at the same time numerous new puncta and striae became apparent.

General examination showed hypertrophic congested papillae and telangiectases on the posterior third of the tongue. The abdominal and thoracic organs were normal.

Laboratory Tests: The Kahn and the Meinicke test were negative. Urinalysis gave negative results, and filarias were not present in the blood on repeated examination. The blood count showed, on July 24, 1937, 70 per cent hemoglobin, 3,920,000 red cells, and 9,000 white cells, with 2 per cent eosinophilis, 54 per cent polymorphonuclears, 24 per cent lymphocytes and 20 per cent monocytes, on August 25, 75 per cent hemoglobin, 3,780,000 red cells and 8,000 white cells, with 8 per cent eosinophils, 70 per cent polymorphonuclears, 20 per cent lymphocytes and 2 per cent mononuclears; on November 12, 65 per cent hemoglobin, 3,650,000 red cells and 16,000 white cells, with no eosinophils, 66 per cent polymorphonuclears, 24 per cent lymphocytes and 10 per cent monocytes.

The coagulation time ranged from two to two and one-half minutes on several determinations; the bleeding time was one minute, and retraction of coagulum, normal. The arm band compression test after five minutes showed a few purpuric spots, which did not increase after ten minutes. The compression test showed anemic halos around the telangiectases and the puncta pronounced, increasing to as much as 3 mm. in diameter.

CAPILLAROSCOPIC DATA

Capillaroscopic examination was performed on the subjects from families 1 and 2 by Dr. Martinez Cañas. In both cases the surface capillaries appeared in excessive numbers, elongated, tortuous and ending in convoluted arches.

HISTOLOGIC OBSERVATIONS

Biopsies were performed in all 3 cases studied, and the pathologic structure was practically identical in all. The epidermis was normal except for slight atrophy corresponding to the most superficial dilated vessels. The papillary layer was edematous, and the connective tissue appeared lax. In the papillary layer as well as in the reticular layer there were numerous dilated vessels of different types. Some consisted of a well formed wall, and some were newly formed capillaries with a single row of endothelial cells; some vessels were limited by a partial endothelial wall, the rest being connective tissue fibers constituting rather vascular lakes in

the surrounding connective magma. Around all these vessels there was cellular proliferation consisting of large ill stained cells of the adventitial type with small nuclei, of numerous lymphocytes and of a few plasma cells. Staining by the Weigert method showed absence, diminution or fragmentation of the elastic fibers in the vessel walls and in the surrounding spaces. The connective tissue around the vessels was loosely woven and edematous. The deeper vessels of the corium were also enlarged and dilated, but the perivascular proliferation was less marked. Where vessels were massed together the microscopic picture was that of angioma.

COMMENT AND CONCLUSIONS

Three families suffering from hereditary multiple telangiectases, a total of 34 members, of which 24 were said to be affected, have been studied. Of the latter, 7 were seen by us, and 3 were made the subject of this study. Of the patients examined, 6 were female and 1 was male. Of the total of 24 persons affected, 13 were female and 11 male. All were white persons, and their ages varied from 4 to 60 years. Actual hemorrhages or a history of hemorrhages together with telangiectasia of the skin and mucous membranes were found in only 3 cases; telangiectases without hemorrhages were found in 19 cases and repeated epistaxis without apparent telangiectases was present in 2 cases. In only 1 case were the repeated hemorrhages severe enough to cause any concern and produce secondary anemia.

Pathologic examination showed rarefaction of the connective and elastic tissue surrounding the vessels of the corium and a proliferation of reticuloendothelial cells and lymphocytes around the vessels, resembling the picture of angioma.

Capillaroscopic study showed excessive richness of surface capillaries, lengthening and tortuosity of the loops and convolution of the terminal arches.

The presence of anemic halos around the capillary dilatations, possibly caused by the rarefaction of the connective tissue, is to be emphasized. The injection of 1 mg. of epinephrine hydrochloride subcutaneously caused delayed dilatation of the blood vessels, occurring between one and two hours after the injection, numerous new capillaries becoming apparent. The anemic halos were made more marked by epinephrine hydrochloride, exposure to cold air and emotional states.

From these observations it may be concluded that hereditary multiple telangiectasia is a congenital, hereditary, familial condition due to a malformation of the capillaries and of their surrounding supporting tissues, with proliferation of the adventitial cells. This condition is similar to angiomatosis. It may also be concluded that it is not primarily and necessarily hemorrhagic and that hemorrhages are probably due to the fragility of the small vessels which lack true walls under increased pressure or small trauma.

KAPOSI'S VARICELLIFORM ERUPTION

A REPORT OF FIVE CASES, IN ONE OF WHICH THE CONDITION BEGAN AS HERPES ZOSTER

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Persons subject to atopic eczema may present a varioliform eruption as a complication. Kaposi [1] recognized this fact, describing the eruption in 1887. Juliusberg [2] also recorded the condition, under the title of pustulosis acuta varioliformis. Both terms suggest a kinship to the eruptive virus diseases, and the presence of the umbilicated pustules certainly adds weight to the possibility of this relationship. The appearance of the firm umbilicated pustule with the dry adherent serosanguineous crust in the umbilication is distinctive; once seen the eruption is not readily confused with those of ordinary pustular dermatoses. The pustules are discrete, and peripheral extension, as encountered in the impetiginous processes, is seldom found. Few of the case reports noted any tendency toward grouping of the lesions, and the eruption was localized most often on the face and neck. The inflammatory reaction is severe; regional adenopathy is the rule, and the systemic phase, indicated by fever, malaise and toxemia, is out of all proportion to the visible cutaneous damage. Early writers held that the process was limited to children, but Goeckerman and Wilhelm [3] McLachlan [4] and Frühwald [5] reported cases of adults, and the present report includes cases of 2 adults, 1 a woman of 70 years. In the reported cases the condition has generally been sporadic in origin and free of any contagious factor; however, McLachlan and Gillespie [6] reported 16

1. Kaposi, M. J.: Pathologie und Therapie der Hautkrankheiten, Vienna, Urban & Schwarzenberg, 1887, p. 483.

2. Juliusberg, F.: Ueber Pustulosis acuta varioliformis, Arch. f. Dermat. u. Syph. **45**:21, 1898.

3. Goeckerman, W. H., and Wilhelm, L. F. X.: Kaposi's Varicelliform Eruption: Report of a Case, Arch. Dermat. & Syph. **32**:59 (July) 1935.

4. McLachlan, A. D.: Kaposi's Varicelliform Eruption, Brit. J. Dermat. **46**: 8, 1934.

5. Frühwald, R.: Pustulosis vacciniformis acuta beim Erwachsenen, Dermat. Wchnschr. **99**:922, 1934.

6. McLachlan, A. D., and Gillespie, M.: Kaposi's Varicelliform Eruption: An Epidemic of Sixteen Cases, Brit. J. Dermat. **48**:337, 1936.

cases that developed in rapid succession in the pediatrics section of a Scottish hospital. Corson and Ludy [7] reported 3 cases of children; they stressed the resemblance to the virus dermatoses but pointed out that one of their patients had had chickenpox and also a successful vaccination. Freund,[8] studying the condition in a 2 year old girl, was able to produce Guarnieri's corpuscles in the cornea of a rabbit inoculated with vesicle fluid and concluded that a virus was the cause. This patient was not immune to vaccination at a later date. Under the caption of eczema vaccination, Ellis [9] reported an eruption developing in eczematous twins who had been associating with an older brother who had an active vaccination take. One twin died. The clinical description and photographs in these cases closely resemble those of cases reported under the term Kaposi's varicelliform eruption.

REPORT OF CASES

CASE 1.—K. Z., a robust white man aged 32, had generalized atopic eczema when first seen in 1934 and again one year later. A papulopustular eruption consisting of discrete firm umbilicated pustules appeared suddenly and symmetrically on eczematous skin of the face and neck. This was accompanied by prostration, malaise and temperatures of 103 to 104 F., and the submaxillary and cervical glands were enlarged. Hospitalization was necessary. The eruption, which was resistant to the usual measures against pyoderma (treatment with ammoniated mercury, potassium permanganate and aluminum acetate), responded slowly to continuous warm wet boric acid dressings. A special nurse and eleven days of hospitalization were required. Recovery was complete, without scarring. Atopic eczema reappeared later.

CASE 2.—J. R., a boy aged 19 months, had had infantile eczema for several months. An acute papulopustular eruption developed suddenly on the face, with severe edema and involvement of the right eyelid. The lesions were discrete shotty umbilicated pustules, with marked surrounding inflammation. Slight submaxillary adenopathy was present, and the temperature was 99 to 100 F. Culture of pustular contents showed Staphylococcus aureus nonhaemolyticus. The child was hospitalized, and the process cleared under continuous warm wet boric acid dressings. Involution was complete, with no scarring.

CASE 3.—J. A., a girl aged 6 months, seen in consultation with her pediatrician, had moderately extensive infantile eczema with a severe papulopustular process involving the face and neck, scalp and extremities. Pustules were numerous, large, firm and umbilicated and arose from inflammatory bases. Edema accom-

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7. Corson, E. F., and Ludy, J. B.: Kaposi's Varicelliform Eruption: Report of Three Cases, Am. J. Dis. Child. **50**:1476 (Dec.) 1935.

8. Freund, H.: Zur Aetiologic der Pustulosis vacciniformis acuta (Kaposi-Juliusberg), Dermat. Wchnschr. **98**:52, 1934; abstracted, Wise, F., and Sulzberger, M. B.: Year Book of Dermatology and Syphilology, Chicago, The Year Book Publishers, Inc., 1934.

9. Ellis, F. A.: Eczema Vaccinatum: Its Relation to Generalized Vaccinia; Report of Two Cases, J. A. M. A. **104**:1891 (May 25) 1935.

panied the dermatitis, and a heavy, cheeselike crust was present on the most greatly involved sectors. There was moderate cervical adenopathy. The temperature was 104. The child's mother, a trained nurse, carried out the therapy of continuous warm wet boric acid dressings. The eruption increased in severity for two days and then began to improve. However, the child's condition became worse. Otitis media developed, the temperature ranged from 105 to 106 F., and the child died eight days later with severe toxemia and probably with pneumonia. Autopsy was not performed. Cultures of material from the pustules showed Staphylococcus albus and cultures of the blood a nonhemolytic streptococcus. The culture of the spinal fluid yielded no organism.

CASE 4.—G. V., a boy aged 3 years, had been seen previously for atopic eczema. Several months later an acute pustular eruption developed on the face and neck. The pustules were of the firm umbilicated type noted in the previous cases and were accompanied by severe edema, cervical adenopathy and moderate elevation of temperature. The services of the nurse who had been on duty in case 1 were secured, and under continuous wet dressings of boric acid solution recovery was secured in four or five days. There was no scarring.

Photograph (case 5) showing the extent of the eruption of umbilicated pustules. Note the zosteriform eruption on the abdomen.

CASE 5.—M. M., a white woman aged 72 years, was first seen as an ambulatory patient. She was highly nervous and presented atopic eczema of seborrheic distribution. On the right flank there was a firm tumor mass about the size of a lemon. It was dull red, doughy, infiltrated and elevated. The surface of this lesion contained a number of irregular dry necrotic ulcerations. Tenderness and pain were severe. The temperature at this time was 99 F. There was no adenopathy, and the spleen was not palpable. History indicated eczema of four months' duration, which had improved and then recurred. Rest in bed, wet dressings, phenobarbital and roentgen rays in fractional doses were given.

Two days later a house call revealed that the patient had been irrational and uncooperative in treatment. A second group of necrotic vesicles had developed anterior to the original lesion, and in addition a number of discrete small umbilicated pustules were scattered at random over the trunk. The temperature was 101 F.

Hospitalization was effected, and the subsequent course was stormy. The temperature rose to 103 F. and fluctuated between 100 and 105 F. thereafter. It

was necessary to confine the patient in bed, restlessness and delirium being out of proportion to the fever. Pain on the right side of the chest was severe. The typical large tense umbilicated pustules had become generalized by the second day in the hospital, and they remained so. The pustules were singularly unresponsive to treatment, and no areas showed any tendency toward involution. Signs of bronchopneumonia developed on the fifth day in the hospital, and death occurred on the ninth.

The urine showed a faint trace of albumin on several occasions. A trace of sugar was present during the first few days only. The blood contained 84 per cent hemoglobin and 5,900,000 red cells, 16,450 white cells, 90 per cent polymorphonuclears, 6 lymphocytes, 2 large monocytes and 2 eosinophils per cubic millimeter. Four days later the hemoglobin content was 87 per cent; the red cells numbered 4,940,000 and the white cells 12,000, with 83 per cent polymorphonuclear leukocytes. Hemolytic Staph. aureus and a hemolytic streptococcus were isolated from contents of a pustule. Blood culture produced a hemolytic streptococcus. (The specimen, of necessity, was drawn through contaminated skin and may not have been reliable.) Culture of blood taken from the heart at autopsy was sterile. The blood was of type II (Moss). Wassermann and Kahn reactions of the blood were negative. The blood contained 141 mg. of sugar per hundred cubic centimeters.

Treatment included administration of an ointment containing 2 per cent ammoniated mercury and an oily lotion, freely applied, morphine sulfate and paraldehyde. Eighty grains (5.17 Gm.) of sulfanilamide was given daily by mouth for three days. Sherman's stock vaccine, iron cacodylate, caffeine with sodium benzoate and a blood transfusion (300 cc.) were given. It was impossible to apply wet dressings because of the patient's extreme restlessness. Later, when the patient was stuporous, dilute solution of sodium hypochlorite U. S. P. (Dakin's solution) was applied experimentally to one leg and seemed to be of value.

Pathologic Report.—A complete autopsy was performed by Dr. Frederick A. Hemsath. Several posterior roots and ganglions were dissected out, together with segments from the right side of the spinal cord corresponding with the level of the zosteriform lesions on the right flank.

Briefly summarized, the pathologic report, besides revealing bronchopneumonia as the immediate cause of death, contained the following histologic data:

Sections of the skin were unsatisfactory because of the technical difficulty of preparation.

Sections of the spinal cord showed no infiltration of the posterior roots in the lower lumbar segment which was studied. In the section of the root and ganglion alone, there was rather intense infiltration of the ganglion with small mononuclear cells. There was also an increase in the interstitial cells of the ganglion. The ganglion cells of the posterior root showed rather severe degeneration.

Within the spinal cord were scattered small grapelike areas of degeneration, in which myelin sheaths and axis-cylinders were lost. These areas were observed in the lateral white columns, in the anterior columns and also in the posterior columns. Myelin sheath stains showed them to be cleancut areas of demyelination.

SUMMARY AND CONCLUSIONS

Five cases of Kaposi's varicelliform eruption are reported.

This complication of atopic eczema is serious, especially in the very young or in the aged.

There were 2 deaths, and all patients required hospitalization or the care of a trained nurse at home.

The condition in 1 case bore resemblance to atypical herpes zoster, and pathologic changes of the nerve and cord also suggested this condition.

The usual measures against pyogenic infections were valueless, and the best results were obtained with continuous warm wet boric acid dressings. It is believed that dressings of dilute solution of sodium hypochlorite U. S. P. would also have been of value.

Recovery is not followed by scarring.

In this group of cases the condition developed sporadically, without contagious or epidemic factors.

SHEEP POX INFECTION IN MAN

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Because of the meager reports in the literature, a case of accidental infection in man by the virus of sheep pox is reported.

One recent article has reported 5 cases in England.[1] No cases have been reported in the United States.

A B

A, Lesion of sheep pox, showing hemorrhagic center and bullous border. The discoloration around the lesion is due to medication. *B*, Lesion after treatment, showing almost complete healing at the border. Note the abortive lesion below the original site.

The lesion is characteristic in appearance,[1] starting as a hemorrhagic pustule, around which a deep bullous border rapidly develops. This results in an umbilication.

The lesion resembles a fresh cowpox vaccination, and the clinical course is similar to that of a vaccination. However, the lesions of sheep pox heal with scarring.

1. Peterkin, G. A. G.: Occurrence in Humans of Contagious Pustular Dermatitis of Sheep ("Orf"), Brit. J. Dermat. **49**:492 (Nov.) 1937.

REPORT OF A CASE

Z. M., a white man aged 32, presented himself because of a single hemorrhagic pustule 3 cm. in diameter on the flexor surface of the right wrist. The border consisted of a deep bleb, which contained blood-stained clear fluid.

The history was definite. He had assorted and delivered 2,000 head of karakul sheep from California and had sustained numerous scratches from burs on his legs and one scratch on his wrist. His hands were protected by gloves.

Three days after he sustained the scratch on the wrist a pustule developed at the site. This increased in size during the following six days. There was transient axillary adenopathy, which lasted two days.

The physical examination disclosed no abnormalities except the poxlike lesion on the right wrist (fig., *A*).

The patient was not acutely ill; the temperature was normal, and culture of material from the lesion yielded only staphylococci. Laboratory examination for sporotrichosis, anthrax and tularemia gave negative results. The blood count was normal. Serologic tests of the blood for syphilis were negative.

The bullous border was clipped off and the lesion painted with 5 per cent methylrosaniline (gentian violet) solution daily.

The patient was hospitalized and given 60 grains (3.88 Gm.) of sulfanilamide daily for three days, without any dramatic effect on the slowly healing lesion.

Healing was almost complete three weeks later (fig., *B*).

SUMMARY

The first case in the United States of sheep pox infection in man is reported.

The clinical appearance, the history of exposure and the course of the disease were typical.

The virus causing the infection is probably different from the virus of true cowpox.[2]

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2. Bonnevie, P.: "Milkers Warts": Infection from "False Cowpox" with a Paravaccinal Virus, Brit. J. Dermat. **49**:164 (April) 1937.

Clinical Notes

RECURRENT LYMPHANGITIS TREATED WITH SULFANILAMIDE

S. R. Mercer, M.D., Fort Wayne, Ind.

The object of this report is to record a case of severe chronic recurrent lymphangitis (elephantiasis nostras) of the upper lip which responded to sulfanilamide therapy. It is my opinion that the aforementioned drug is solely responsible for the improvement. No attempt will be made to review the literature or comment on other means of treatment, aside from mentioning those previously employed in this case. The observations to be reported were purely clinical throughout. No untoward reaction was noted from the use of the drug during or after its administration.

REPORT OF A CASE

Miss M. S., aged 29, was first seen on June 14, 1937, on account of enlargement and recurrent swelling of her upper lip. She stated that one day in March 1935, after swimming in an indoor pool, her upper lip became red and irritated. A severe cold followed by a sinal infection (left antrum) ensued. Local treatment and injections of stock vaccine served to clear the sinal infection in two months, but the enlargement of the upper lip which had taken place persisted. A loss of 10 pounds (4.5 Kg.) took place in that period. From then on until 1937 she had recurrent attacks of inflammation and swelling of the upper lip, and at one time her entire nose was involved. Each time the acute inflammatory process would subside, only to leave the lip slightly larger. The severest exacerbations took place when a cold was contracted, but there were exacerbations too (to a lesser degree) on exposure to paint fumes, strong sunlight or rainy weather and several days previous to her menstrual periods.

Several complete physical and dental examinations were made during 1935 and 1936. No particular pathologic conditions could be found. Numerous laboratory procedures were employed from time to time without avail. Medication by mouth, administration of vaccines (stock and autogenous), application of unfiltered roentgen rays locally, cutaneous tests, elimination diets, changing cosmetics and pillows, ultraviolet irradiation (generalized) and chiropractic "adjustments" had been tried.

Administration of 20 grains (1.29 Gm.) of sulfanilamide per day was begun on June 21, 1937. Medication was continued until the last week in August, when she stopped treatment on her own volition. During that period regression of the lesion was noted in spite of several minor flare-ups, the worst one being due to a prolonged exposure to the sun. On September 5 a cold developed and with it a severe exacerbation of the lesion. Sulfanilamide therapy in the same dosage was resumed at that time and continued for two months, followed by an equal period of rest, and then resumed for another two months, for a total of six months' ingestion out of a possible eight. A course of stock streptococcic vaccine was then administered. No improvement was noted from this form of therapy, and as it was thought that maximum improvement had been obtained, all medication except administration of an iron preparation was discontinued.

At the present time the lip is conservatively estimated to be one-sixth to one-fifth the size it was when first seen and is compatible with the facial contour. Since the withdrawal of medication the patient has had only the slightest transient "puffing" of the lip from time to time, and that only when a cold has been present.

Considering the original size of the lip, the severity of the exacerbations and the duration of the enlargement, the result has been excellent, and the mental outlook and general physical condition of the patient have been improved immeasurably.

An added note of interest is that the patient spent two weeks in Miami, Florida, in March 1939, where she took numerous sun baths, without any disturbance of the lip.

SUMMARY

A case of recurrent lymphangitis (elephantiasis nostras) of the upper lip greatly benefited by prolonged sulfanilamide therapy is reported.

315 Wayne Pharmacal Building.

USE OF MAPHARSEN FOR TERMINATING MALARIA ARTIFICIALLY PRODUCED BY INOCULATION

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Dermatologic Consultant
AND
S. E. C. Turvey, M.D.
Neurologic Consultant
VANCOUVER, BRITISH COLUMBIA, CANADA

The use of mapharsen to terminate malaria artificially produced by inoculation in the treatment of neurosyphilis in a series of 24 cases was recently reported by Goldman.[1] While it was recommended that to insure against recurrences three or four injections should be given, it was stated that in over 90 per cent of the cases a single injection sufficed to terminate the malaria permanently.

As a result of our experience in treating patients inoculated therapeutically with the organisms of tertian malaria, as were Goldman's, we are heartily in accord with his recommendation that three or four injections should be given to insure against recurrences We are inclined to believe that more are advisable.

In a series of 9 consecutive patients treated by the Section of Venereal Disease Control of the British Columbia Provincial Board of Health in Vancouver, one injection of 0.04 to 0.06 Gm. of mapharsen was given, the dose varying with the patient's weight.

Four of these patients (44 per cent) had no recurrence. Five had two or more chills after the injection. Ont of these, a woman, had high fever with rigors, and the organism was recovered from the blood.

While mapharsen may supplant quinine as a means of terminating malaria, we should recommend, especially in view of its value and constant therapeutic

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From the Section of Venereal Disease Control, British Columbia Provincial Board of Health, Donald H. Williams, M.D., Director.

1. Goldman, D.: The Use of Mapharsen in the Treatment of Malaria, Am. J. M. Sc. **196**:502 (Oct.) 1938.

index in syphilis, that a full course of injections of mapharsen be given in place of the quinine ordinarily used and the course of neoarsphenamine with which the termination of the malaria is usually followed.

A single dose of mapharsen should not be relied on for arrest of the course of therapeutic malaria, especially where it is customary to permit the patient to return home for a two weeks' recuperation period, during which time he is not ordinarily seen by the medical attendant, as has been the practice where quinine is used. Our experience has shown, as before mentioned, that when the malaria appears to have been cured it may have been only temporarily arrested.

TREATMENT OF MULTIPLE PIGMENTED HEMORRHAGIC SARCOMA (KAPOSI)

LESTER HOLLANDER, M.D., AND JOSEPH M. SHELTON, M.D., PITTSBURGH

During the past year we have had a case of typical multiple pigmented hemorrhagic sarcoma (Kaposi).

REPORT OF A CASE

The patient, G. C., a Swede aged 67, presented on both lower extremities numerous firm bluish rounded nodules varying in size from that of a split pea to that of a bean, of one year's duration. Histologic examination confirmed the clinical diagnosis of multiple pigmented hemorrhagic sarcoma.

Since we have been using contact roentgen irradiation for intraoral and certain selected superficial cancers, we decided to treat several of these lesions in the same manner. Treatment was directed to one of the largest nodules. A single dose of 1,620 roentgens was given. The factors used were 50 kilovolts, 4 milliamperes, 3 cm. target-skin distance and time six minutes. The inherent filter factor of our contact therapy tube is approximately 0.2 mm. of nickel. (We have found the half value layer to be approximately 1 mm. of aluminum.)

In about ten days a moderate erythema was present around the treated lesion, and during the ensuing four weeks the nodule completely receded. It has not recurred during the past year.

Since then eight additional lesions have been treated with single doses varying from 600 to 1,650 r. It was found that these lesions required a minimum dose of 1,500 r; all lesions treated with this dose have completely receded.

We think that this method of therapy is particularly well adapted for the treatment of this entity, on account of the ease of application and the ready response of the lesions.

The optimum dose of roentgen rays has been established at 1,500 r.

From the Pittsburgh Skin and Cancer Foundation.

Abstracts from Current Literature

EDITED BY DR. HERBERT RATTNER

MONOCYTIC LEUKEMIA AND OTHER NEOPLASTIC DISEASES OCCURRING IN MICE FOLLOWING INTRASPLENIC INJECTION OF 1:2-BENZPYRENE. J. FURTH and O. B. FURTH, Am. J. Cancer **34**:169 (Oct.) 1938.

After intrasplenic injection of 1,2-benzpyrene, a carcinogenic agent, in 96 mice, 9 cases of monocytic leukemia, 2 cases of neoplastic hemangioma and 2 of atypical sarcoma were observed. These conditions are rare in mice of the stock used in these experiments and as controls. The incidence of pulmonary tumors and of myeloid leukemia was considerably increased in the treated group of mice.

OCCURRENCE OF CANCER IN RATS TREATED WITH OESTRONE. C. S. MCEUEN, Am. J. Cancer **34**:184 (Oct.) 1938.

Histologically malignant tumors, chiefly squamous cell carcinoma, were observed more frequently in a group of rats treated for long periods with estrone and subjected to local irritation, such as that produced by gashouse tar, than in a control group.

HODGKIN'S DISEASE WITH ULCERATIVE INVOLVEMENT OF SKIN. S. B. PESSIN and ERNST POHLE, Am. J. Cancer **34**:220 (Oct.) 1938.

The authors report a case of ulcerative lymphogranuloma of the skin of Hodgkin's type, which they considered to be secondary to primary osteomyelitic Hodgkin's disease of the underlying sternum. The lesion healed after roentgen therapy, but three years later multiple cutaneous ulcers and generalized lymphadenopathy developed, terminating in death.

LEUKEMIC LYMPHOBLASTOMA IN CALF: HEMATOLOGIC AND HISTOLOGIC STUDY. J. STASNEY and W. H. FELDMAN, Am. J. Cancer **34**:240 (Oct.) 1938.

This study indicated that the immature lymphocytes in the peripheral blood stream originated as immature cells of the reticulum cell type in involved lymph nodes. The histologic picture of involved tissues and the peripheral blood stream was that of lymphatic leukemia in man. Some of the enlarged lymph nodes showed the histologic picture of lymphosarcoma.

FOERSTER, Milwaukee.

CLINICAL ASPECTS OF ANEURYSM. JOHN H. MILLS and BAYARD T. HORTON, Arch. Int. Med. **62**:949 (Dec.) 1938.

Mills and Horton record the incidence of syphilis in a series of 596 cases of aneurysm. When the cases were classified according to the location of the aneurysm, syphilis was found in 3.5 per cent with intracranial aneurysm in 70 per cent with thoracic disease, in 9 per cent with abdominal involvement and in 9 per cent with involvement of the extremities. In the 339 cases of thoracic aneurysm, the ratio of male to female patients was approximately 4 to 1. Arteriosclerosis was thought to be an important factor in the development of abdominal aneurysm and of thoracic aneurysm in older persons. Aneurysm of the peripheral arteries occurs earlier in life than aneurysm of other parts of the body.

LYNCH, St. Paul.

MICROSCOPIC AGGLUTINATION TEST FOR DIAGNOSIS OF SWINE ERYSIPELAS. A. G. KARLSON and S. H. MCNUTT, J. Infect. Dis. **64**:49 (Jan.-Feb.) 1939.

The authors describe a microscopic test which they found more reliable than the tube test previously described. Directions are given for preparing an inexpensive medium to obtain antigen and for routine work with the organism of swine erysipelas. Dilutions of the serum are made in watch glasses. Hanging drops are then made from each dilution, and each slide is subjected to incubation for fifteen minutes. Examination of the preparations with the low power lens will reveal clumping of the bacteria when the test is positive. When normal serum is used the bacilli will remain uniformly distributed throughout the field. Checks or controls are essential.

ACTION OF PRONTOSIL-SOLUBLE AND SULFANILAMIDE ON PHAGOCYTIC ACTIVITY OF LEUKOCYTES AND ON DISSOCIATION OF STREPTOCOCCI. RUTH TUNNICLIFF, J. Infect. Dis. **64**:59 (Jan.-Feb.) 1939.

The author found that neoprontosil (formerly prontosil soluble) and sulfanilamide in dilutions up to 1:1,000,000 for the former and to 1:2,000,000 for the latter in saline solution promote phagocytosis of greening and hemolytic streptococci. Both preparations appear to stimulate the activity of leukocytes but do not act as opsonins. Stabilized R cultures of greening streptococci are more phagocytable than their stabilized S forms. Streptococci grown in from 1 to 10 per cent neoprontosil or in 1:1,000,000 sulfanilamide in 1 per cent dextrose broth show by chain formation and granular growth some S→R change which appears to make the streptococci more phagocytable. A 1:1,000 solution of neoprontosil and a 1:1,000,000 solution of sulfanilamide promote phagocytosis of streptococci in pus. Neoprontosil and sulfanilamide injected subcutaneously into 9 patients with monocytic leukemia and 2 with neoplastic hemangioma appear to have stimulated the activity of the leukocytes to the same degree.

CORNBLEET, Chicago.

EFFECTS OF INTRAMUSCULAR INJECTIONS OF VITAMIN B_1 ON ACUTE LEPROUS NEURITIS AND OF ORAL ADMINISTRATION ON GENERAL DISEASE: PRELIMINARY REPORT. L. F. BADGER and D. W. PATRICK, Pub. Health Rep. **53**:969 (June 17) 1938.

During the six months preceding their report Badger and Patrick treated 10 patients who had severe acute leprous neuritis of the peripheral nerves with intramuscular injections of thiamin chloride. The injections were begun as soon as possible after the onset of symptoms, or when the patient first complained of pain. The procedure followed was administration of 300 international units once a day by intramuscular injection and twice a day by mouth. Of the 7 cases in which the injections were begun on the day of onset, the pain disappeared completely twenty-four hours after the first injection in 4, in forty-eight hours in 1 and on the fourth day in another. In 1 case, owing to required emergency treatment, the injections were discontinued after two had been given; on the third day the pain was moderate, after which it continued to lessen in severity until the seventh day, when it was no longer present. Tenderness could no longer be elicited after twenty-four hours in 1 case and after three or four days in all but the 1 case in which the treatment was interrupted. In this case no tenderness could be elicited on the seventh day. In each case the tenderness was less marked twenty-four hours after the first injection. Definite diminution in the swelling occurred about the time that the tenderness disappeared. In 1 case the treatment by injection was begun on the second day of symptoms, and twenty-four hours later the pain lessened definitely. The pain disappeared entirely after three injections, and after four injections the tenderness disappeared completely. In 2 cases the injections were begun on the fourth and fifth days of symptoms. In one of these the response

to treatment was not as abrupt, and the improvement was not sharply defined from day to day. In the other case the results were about as prompt as in the majority of the cases.

J. A. M. A.

HEREDITARY TRANSMISSION OF LYMPHOGRANULOMATOSIS VENEREA. WALDEMAR E. COUTTS and OLGA MONETTA, J. Trop. Med. **41**:279 (Sept. 1) 1938.

In 1936 Dick cited a case of unusual interest, that of an infant 2 weeks of age with a positive Frei test. Both the father and the mother were known to have lymphogranuloma venereum. Dick was of the opinion that intrauterine infection had occurred.

The authors cite one of their own cases in which a mother who had a severe form of the disease (positive Frei test) delivered an infant whose Frei test on the second and third days of life was negative. Examination of the placenta showed no lymphogranulomatous lesions.

The authors feel that the question of the hereditary transmission of lymphogranuloma venereum should be studied further and that the scanty data at hand permit no definite conclusions.

LAYMON, Minneapolis.

ACUTE IODIDE ERUPTION FOLLOWING INGESTION OF SMALL AMOUNT OF POTASSIUM IODIDE. IVAN M. WARTZKI, M. J. Australia **2**:738 (Oct. 29) 1938.

A case of vesicopustular and bullous iodide eruption followed by death is described in a 66 year old man with advanced cardiac and renal disease. It occurred as a result of the ingestion of a small amount of potassium iodide, three doses of 0.3 Gm. each.

The question arises whether the man had a peculiar idiosyncrasy to iodides which may not be explained by the defective elimination. Death may have been due, at least in part, to the constitutional disturbances accompanying the eruption.

A warning is sounded regarding the indiscriminate use of iodides in cases of cardiac and renal disease.

BAER, Pittsburgh.

COMMENT CONCERNING QUESTION OF ERYTHEMA NODOSUM. L. M. PAUTRIER, Bull. Soc. franç. de dermat. et syph. **45**:1046 (July) 1938.

Pautrier states that he discussed the subject of erythema nodosum at great length with Debré, who defended the theory of internists and pediatrists, that tuberculosis is the predominant and perhaps the sole etiologic factor, while Pautrier held to the view of most dermatologists, that erythema nodosum is a symptom complex due to multiple causes. Pautrier explained the differences in opinion in the following way: Erythema nodosum seen by the pediatrist in infants has a tuberculous background in almost all cases, whereas the disease in adults, studied most frequently by the dermatologists, may be an accompaniment of diverse infections, such as syphilis, lepra and trichophytosis.

Pautrier believes that erythema nodosum and erythema multiforme in all probability possess the same pathogenesis, that they may represent a cutaneous sensitization, in different planes, to the same etiologic agents. He mentions the occurrence of erythema multiforme in 5 of 26 cases of erythema nodosum.

ERYTHEMA NODOSUM IN INFANT. E. LESNÉ, Bull. Soc. franç. de dermat. et syph. **45**:1086 (July) 1938.

From 1920 to 1938 the author observed 96 cases of erythema nodosum, which he regards as a definite sign of a primary tuberculous infection in the infant, equaling in importance the tuberculin reaction or phlyctenular conjunctivitis. In 50 per cent of the cases it was possible to find a source of tuberculosis within the family. In 95 of the 96 cases the tuberculin reaction became positive during the course of the eruption.

ERYTHEMA NODOSUM AND TUBERCULOSIS. ROBERT DEBRÉ, Bull. Soc. franç. de dermat. et syph. **45**:1091 (July) 1938.

The author states that during the past several years he has observed more than 150 cases of erythema nodosum, although only 50 were used as a basis for the present study. To Debré erythema nodosum is always tuberculous. In half the cases roentgenograms showed shadows diagnostic of a primary pulmonary infection. In about half the cases tubercle bacilli were discovered in the sputum or gastric contents, even without characteristic roentgen evidence in the chest. In 2 cases positive evidence of tuberculosis was obtained by inoculating tissue from the cutaneous nodules into monkeys. Tubercle bacilli were found in the blood in 4 cases. A source of tuberculosis was traced in about half the cases. The seasonal nature of erythema nodosum corresponds to similar seasonal variations in tuberculous pleurisy and meningitis. Debré believes that erythema nodosum is not an allergic manifestation of tuberculosis but an inflammatory lesion depending on the actual presence of tubercle bacilli in the nodules.

ETIOPATHOGENIC STUDY OF THIRTY-EIGHT CASES OF ERYTHEMA NODOSUM. E. RAMEL, Bull. Soc. franç. de dermat. et syph. **45**:1138 (July) 1938.

In a long article the author expresses his views concerning erythema nodosum. His conclusions are as follows:

1. So-called idiopathic erythema nodosum is always a cutaneous manifestation of tuberculous bacillemia, in adults as well as infants. There is usually a primary infection, although in adults there may be a superinfection. From a biologic point of view erythema nodosum is a tuberculid—that is, an allergic manifestation produced by the hematogenous propagation of tubercle bacilli of modified virulence.

2. Symptomatic or secondary erythema nodosum is to be divided into two classes: (*a*) one clinically no different from the idiopathic type, with a tuberculous background and brought about by a biotropic mechanism like that occurring in syphilis or following childbirth; (*b*) one of toxi-infectious origin, homologous with true erythema nodosum but distinguishable by its etiologic agents (various bacteria, toxins and fungi). The terms erythema nodosum and dermatitis contusiformis should be reserved for only the first type, while eruptions of the other type should be designated as "id" eruptions (nodose trichophytids and the like).

ERYTHEMA NODOSUM AND POLYMORPHOUS ERYTHEMA. H. GOUGEROT, Bull. Soc. franç. de dermat. et syph. **45**:1195 (July) 1938.

After a long discussion Gougerot concludes that polymorphous erythema in its various types (purpuric, erythematous, papular, nodular, nodose) is a syndrome related to different bacteria (most frequently tubercle bacilli) acting on a sensitized terrain. This view explains the identity of the reactions to various causes, the evolution and the good prognosis, since polymorphous erythema represents a victorious defense reaction.

ACUTE VULVAR ULCER OF LIPSCHÜTZ AS MANIFESTATION OF POLYMORPHOUS ERYTHEMA. LUBIN POPOFF, Bull. Soc. franç. de dermat. et syph. **45**:1254 (July) 1938.

Mucosal localization of erythema multiforme, such as ectodermosis pluriorificialis erosiva, is rare. Acute vulvar ulcer is rare. Bacillus crassus has been shown to be a nonobligatory saprophyte in the latter condition. In many cases of acute vulvar ulcer B. crassus is not present. From his studies the author concludes that acute vulvar ulcer is one of the many manifestations of erythema multiforme and suggests that the latter disease is related to tuberculosis.

LUPUS ERYTHEMATOSUS IN INFANT AGED ELEVEN MONTHS. FERNET P. WEISSENBACH and LE BARON, Bull. Soc. franç. de dermat. et syph. **45**:1681 (Nov.) 1938.

The mother of the infant discussed in the report had been treated for lupus erythematosus at a previous time. Injections of bismuth and gold preparations resulted in complete healing. Later she brought her 11 month old child for examination, and it was discovered that the child too had lupus erythematosus on the face. Injections of a bismuth preparation caused the lesions to disappear. The child showed no evidences of tuberculosis.

TREATMENT OF CHANCROID WITH POWDER OF PARA-AMINO-PHENYL-SULFAMIDE. LEPINAY, Bull. Soc. franç. de dermat. et syph. **45**:1728 (Nov.) 1938.

The author comments on the brilliant results of sulfanilamide therapy of venereal diseases and states that the use of these drugs for chancroid is simple, painless and extremely effective. For early disease the drug is used as a local dusting powder, while for more advanced or severe involvement it is used both locally and by mouth. As a general rule lesions heal within a week, although the recent chancroids heal sometimes within three or four days.

HEALING OF PARAPSORIASIS AFTER INJECTIONS OF VITAMIN A. A. TZANCK, E. SIDI and J. P. PAILLAS, Bull. Soc. franc. de dermat. et syph. **45**:1818 (Dec.) 1938.

The patient was a man aged 57. Lesions of parapsoriasis of the plaque type were located on the chest. After a dozen intramuscular injections of vitamin A the eruption involuted.

LAYMON, Minneapolis.

KONDO PRECIPITATION REACTION IN SYPHILIS. Y. IGA and M. TOMATU, Lues: Bull. Soc. japon. de syph. **17**:14 (June) 1938.

In the Kondo precipitation test inactivated serum of the patient and cholesterolized extract of the heart of a plant-eating animal are used. The result may be read in one and one-half to two hours. It coincides in 90 per cent of cases with the result of the Wassermann and the Murata test.

RESULTS OF IDE TEST IN SYPHILIS. K. TEI, Lues: Bull. Soc. japon. de syph. **17**:18 (June) 1938.

The author found that the Ide test, which is a color test, compares well with the Murata and the Wassermann test when whole blood or inactive serum is employed. The test with active serum gives a low percentage of positive reactions and occasionally an unspecific reaction.

PRODUCTION OF THERAPEUTIC RELAPSING FEVER BY ADMINISTRATION OF SPIROCHAETA RECURRENTIS THROUGH MUCOUS MEMBRANES. T. YAMASHITA, Lues: Bull. Soc. japon. de syph. **17**:31 (June) 1938.

The author found that a suspension of Spirochaeta recurrentis administered by rectum in the form of an enema produces high fever and two to seven recurrent attacks of fever after an incubation period of one to three days. For the purpose of fever therapy he recommends this method of administration in preference to the oral or the subcutaneous method.

BY-EFFECTS OF ARSPHENAMINE. S. MATSUMOTO, Lues: Bull. Soc. japon. de syph. **17**:111 (Aug.) 1938.

Among 3,016 patients treated with arsphenamine, eruptions including immediate reactions and early morbilliform and scarlatiniform and late exfoliative eruptions

developed in 112. In some patients jaundice developed simultaneously. An eruption like keratosis follicularis (Darier) developed in 1 patient; in another there was an erythema nodosum-like eruption on both legs, and a third presented erythematous edema of both upper extremities after the fifth injection.

The author considers the present methods of testing the toxicity of arsphenamine in mice not reliable because (1) toxicity in mice does not parallel that in human beings, (2) mice are hypersensitive to an excess of alkalis, and (3) they react differently in the different seasons. He states further that values for the toxicity of arsphenamine determined by tests of hepatic function in rabbits are not reliable for human beings.

The present day prophylaxis and treatment of by-effects of arsphenamine with sodium thiosulfate, dextrose, vitamin C and amino-acetic acid are lacking experimental and clinical justification.

SOME PROBLEMS IN EXPERIMENTAL SYPHILIS. S. MATSUMOTO, Acta dermat. **31**:51 (April) 1938.

The author discusses some problems of experimental syphilis. For the purpose of investigating problems of immunity the methods of reinfection and superinfection are used. The quantitative inoculation method permits the determination of the spirochetal content of the organs. Immunity to syphilis varies according to the different animals used. The author considers "chancre immunity" in rabbits as an expression of acquired immunity and symptomless reinfection as a sign of weak but not absent immunity. The author considers the so-called flaring up reaction in healed inoculation chancres of animals which have been treated and reinfected intravenously with a suspension of spirochetes specific and related to the Shwartzman phenomenon. This reaction may be used to differentiate syphilis from frambesia. It has been observed also after intravenous superinfection.

Inoculation chancres and general eruptions produced in animals are almost identical with those in men. They also respond promptly to antisyphilitic therapy. The animals, however, may harbor spirochetes in the lymph glands and spleen for a long time. No syphilis of the nervous system or congenital syphilis was observed in animals.

Syphilitic manifestations depend on the species, the age of the animal, the dose and method of inoculation and the season. Internal factors may also play a role. Acidosis favors involvement of the bones, while lipoidosis inhibits the inoculation lesion. The author counsels caution in applying the results obtained in experimental syphilis to syphilis in men. Although experimental syphilis in animals has brought out many interesting facts, it has not aided directly the investigation of human syphilis.

Numerous dyes have been found to stain the pale spirochete better and more rapidly than Giemsa stain. It is not possible to differentiate by means of staining between Spirochaeta pallidula and Spirochaeta cuniculi.

It is still doubtful if leukoplakia has any relation to syphilis. In Japan genital precanceroses and cancer of the penis are much more frequently encountered than in other parts of the world. In the majority of cases carcinoma of the penis develops from precancerous lesions. Precancerous lesions develop almost exclusively from a phimotic prepuce.

STAINING OF SPIROCHETES IN SMEARS BY MEANS OF POTASSIUM PERMANGANATE SOLUTION. K. ONO, Acta dermat. **31**:69 (April) 1938.

The author found that the spirochetes of syphilis are stained intensely with a 1 per cent potassium permanganate solution. Fixation of the smear is accomplished best with solution of formaldehyde U. S. P. At room temperature three to seven days is required for staining, or ten to twenty-four hours when the slides are put into an incubator at 60 C.

BLOOM, New York.

FERMENT STUDIES IN BROWN-PEARCE CARCINOMA: I. AMYLASE IN THIS CARCINOMATOUS TISSUE AS WELL AS IN SOME ORGANS (LIVER, KIDNEY, MUSCLE, TESTICLE) AND IN SERUM OF RABBITS BEARING THIS TUMOR. G. MORI, Acta dermat. **32**:1 (Aug.) 1938.

The author studied the amylase content in Brown-Pearce carcinoma of rabbits and its metastases and in the noninvaded organs of the affected rabbits. In the tumor itself the amylase activity was weak in the non-necrotic tissue, more marked in half necrotic tissue and most marked in necrotic carcinoma tissue. The noninvaded liver and kidney did not differ greatly in their amylase content from the organs of noncarcinomatous rabbits. In the advanced and terminal stages, however, an increase in amylase activity was found in the liver and a decrease in the kidney. The presence of metastases in these organs did not change their amylase content. The muscle tissue of carcinomatous rabbits showed a marked decrease of amylase activity starting at the time of the development of the inoculation tumor and increasing continuously with the progress of the tumor. A slight difference of amylase activity was noted in the testicles. Of 25 carcinomatous rabbits, 14 showed a decrease in the amylase activity of the blood serum; this decrease started with the beginning development of the tumor and increased continuously with the greater nutritional disturbance produced by the carcinoma.

INFLUENCE OF ACIDOSIS PRODUCED BY ADMINISTRATION OF DEXTROSE ON SYPHILITIC LESIONS. SEN NAGAI, Acta dermat. **32**:12 (Aug.) 1938.

In rabbits in which acidosis had been produced by administration of dextrose syphilitic inoculation lesions developed much earlier and were more pronounced than in control animals. On the front legs a pea-sized hard tumor developed frequently. After intravenous inoculation of spirochetes the generalized eruption developed much earlier and was more pronounced than on control rabbits. Besides the osseous lesion on the front legs one was noted in the supraorbital region. Earlier and more intense development of syphilitic lesions was noted also after paranasal osseous inoculation.

Society Transactions

AMERICAN DERMATOLOGICAL ASSOCIATION, INC.

JAMES H. MITCHELL, M.D., *President*

Clinical Meeting, San Francisco, June 8, 1938

FRED D. WEIDMAN, M.D., *Secretary*

Epidermolysis Bullosa. Presented by DR. MERLIN TREVOR-ROPER MAYNARD, San Jose, Calif.

B. D., a white girl aged 2½ years, is presented because of vesiculobullous lesions which rupture and leave superficial crusted erosions. They have occurred since birth, mainly about points subject to friction and trauma, such as the tips of the fingers, the elbows and the heels.

She was presented before the San Francisco Dermatological Society on Sept. 27, 1935 (ARCH. DERMAT. & SYPH. **34**:140 [July] 1936). She was treated with local soothing and antiseptic ointments and protective bandages up to Aug. 18, 1936. At this time many severe ulcerative lesions were present on the feet, backs of the legs, abdomen and hands. She then was given enteric-coated tablets of anterior pituitary. On Sept. 1, 1936, she was considerably better. This therapy was continued until September 29, at which time she was relapsing somewhat. She was not seen again until the present time.

Epidermolysis Bullosa. Presented by DR. N. N. EPSTEIN, San Francisco (for the Department of Pediatrics and the Division of Dermatology of the University of California Medical School).

A. B., an Italian baby aged 7 weeks, was in good general condition and weighed 7 pounds 1½ ounces (3,218 Gm.) at birth. The mother is 21 years old, and the pregnancy was uneventful except for mild pyelitis. Labor lasted four hours, and delivery was spontaneous. One sibling is alive and well and has had no cutaneous abnormalities.

Physical examination an hour after birth revealed clubbed feet. On both ankles and feet there were circumscribed sharply defined depressed smooth reddened areas in which there was an apparent lack of skin. The surface consisted of a thin layer of tissue through which could be seen the blood vessels of the underlying subcutaneous tissue. A bulla 1 cm. in diameter was present on the dorsum of the left index finger. It was filled with clear fluid. On the dorsum of the right index finger was evidence of a recently ruptured small bulla. The mucous membrane of the mouth and tongue had sloughed, leaving a raw, reddened surface.

Since birth gradual improvement has occurred. There has been, however, a slough of the distal phalanx of the right great toe. Bullae have also appeared on the elbows, knees, fingers, ears, back and abdomen.

The mother's Wassermann reaction and that of blood from the umbilical cord were negative. The blood count was normal. Roentgenograms showed no abnormalities of the bones of the legs or feet or of the chest or lumbar part of the spine. The thymus was not enlarged.

Dermatitis Herpetiformis (Bullous Dermatosis of Childhood [Bowen]). Presented by DR. H. J. TEMPLETON, Oakland, Calif.

A. H., a white boy aged 9 years, has had a generalized vesiculobullous eruption for over three years. Extensive clinical and laboratory studies, including urinalyses, blood counts, Wassermann tests, examinations of stools, blood cultures and electrolytic tests of blister fluid, have failed to reveal anything significant.

Numerous therapeutic procedures have been tried without apparent result. This patient has been presented twice before the San Francisco Dermatological Society, on April 17, 1936 (ARCH. DERMAT. & SYPH. **36**:464 [Aug.] 1937) and Sept. 18, 1936 (ibid. **36**:658 [Sept.] 1937). Since the onset he never has been entirely free from blebs. However, the lesions are gradually decreasing in number. There has been no great disturbance in the general health or development of the patient.

Dermatitis Herpetiformis (Bullous Dermatosis of Childhood [Bowen]).
Presented by DR. H. J. TEMPLETON, Oakland, Calif.

R. C., a white boy aged 3 years, entered the Alameda County Hospital with a history of an eruption of one week's duration, associated with two attacks of vomiting at the onset. There was no history of other recent illness or of vaccination. The past and family history were unimportant.

Inspection revealed a well developed and well nourished boy, apparently not acutely ill. His body and extremities were covered with crusted lesions varying in diameter from 5 mm. to 2 cm. A few primary lesions, vesicles and blebs, were present. An initial diagnosis of impetigo contagiosa was made, and treatment with methylrosaniline (gentian violet), ammoniated mercury ointment and baths of solution of sodium hypochlorite was prescribed.

There was little change for two weeks; a few new lesions continually appeared. Then suddenly a shower of new vesicles and bullae developed, mostly on the trunk. They at first appeared to arise from normal skin, but a faint erythematous halo developed within twenty-four hours. The contents, at first clear serum, became purulent within twenty-four to forty-eight hours. There was a tendency toward grouping. Apparently there was little pruritus. At present the process is still active on his extremities but has cleared somewhat on the genitals, abdomen and thorax.

He was rather toxic during the second week of observation, but now he feels well. The temperature has ranged from 100 to 102 F. and the pulse rate from 100 to 120. Conjunctivitis developed eleven days after entry, but this has disappeared.

Examinations by the pediatric and otolaryngologic services have disclosed nothing of interest.

Urinalyses have given normal results. Blood counts on two occasions showed 18,500 and 19,400 leukocytes. Differential counts were normal. There was no eosinophilia. Cultures of material from the nose, throat and rectum were normal. Cultures from untreated blebs showed beta hemolytic streptococci and Staphylococcus aureus. The Wassermann reaction was negative.

The administration of sulfanilamide for three days did not alter the appearance of the eruption.

The bullous character of the eruption, its occurrence in childhood, the absence of polymorphism and the relative absence of pruritus conform to the findings of Bowen in his cases of bullous dermatosis of childhood.

DISCUSSION OF BULLOUS ERUPTIONS IN CHILDHOOD

DR. JOSEPH GRINDON, St. Louis: I think A. H. and R. C. have dermatitis herpetiformis. The condition is somewhat different from the ordinary type, but in early childhood the epidermis is not so firmly attached to the cutis as it is later in life. This may account for the much larger lesions.

DR. CLARK W. FINNERUD, Chicago: I have seen several children about the ages of A. H. and R. C. for whom a diagnosis of dermatitis herpetiformis has been accepted by all concerned. The picture differed from that in these cases only in that the lesions were not pustular. The children were extremely high strung. After they had ingested large amounts of solution of potassium arsenite the condition eventually cleared, and the skin and general health were subsequently unimpaired.

DR. FRED D. WEIDMAN, Philadelphia: I believe the condition of A. H. and R. C. has been described under the name hydroa puerorum by the British and

is considered as a modified form of dermatitis herpetiformis. In the case of A. B. a remarkable destruction of tissue has taken place around the feet. The picture of the child in the earlier stage made me think of a forme fruste of Ritter's disease. I noticed in the photographs that there was dermatitis on the face, which is consistent with Ritter's disease. In any event, the condition is not typical.

DR. DONALD M. PILLSBURY, Philadelphia: I have observed a number of cases of this type in the Children's Hospital in Philadelphia. I think that Dr. F. C. Knowles some years ago in an article on dermatitis herpetiformis in children (*J. Cutan. Dis.* **25**:247, 1907) pointed out the fact that in many of these cases the lesions undergo spontaneous involution. Recently in 2 cases of bullous eruptions in children associated with sensitivity to iodides I attempted desensitization with small increasing doses of iodides by mouth, without any success.

DR. H. J. TEMPLETON, Oakland, Calif.: In 1905 J. T. Bowen presented a series of cases of bullous eruptions occurring in children who had been vaccinated shortly before the disease appeared (*J. Cutan. Dis.* **19**:401, 1901). Later he presented another series in which only half of the children had been vaccinated (ibid. **24**:110, 1906). He regarded these postvaccinal bullous dermatoses as variants of dermatitis herpetiformis. He noted certain differences from dermatitis herpetiformis in adults, viz., there is not as much multiformity, the lesions being mostly bullous; there is less pruritus; there is a tendency to spontaneous but slow recovery. Although my patients had not been vaccinated prior to the onset of their eruptions, I believe they present all the criteria for a diagnosis of dermatitis herpetiformis of the Bowen type.

Necrobiosis Lipoidica Diabeticorum. Presented by DR. L. R. TAUSSIG, San Francisco (for the Division of Dermatology of the University of California Medical School).

D. S. P., an American housewife aged 19, was first seen in September 1934, when a diagnosis of diabetes mellitus was made. She had been known to have diabetes since the age of 12 years. From 1934 to 1938 her course has been somewhat stormy, owing to frequent occurrences of coma, for which she has been hospitalized on several occasions.

This patient entered the dermatologic clinic on April 8, 1938, complaining of lesions on her left leg of four months' duration. Examination showed two lesions. The upper was a fairly superficial indurated area about 2 cm. in diameter and somewhat purple. The lower one was about 7 cm. in diameter and consisted of diffuse erythematous induration, duller in the center, with some scaling. It was moderately tender. Between the upper and the lower lesion there was a circumscribed area of slightly tender subcutaneous induration but no change in the skin. Physical examination was otherwise essentially normal.

At present she is on a diet consisting of 150 Gm. of carbohydrate, 40 Gm. of protein and 80 Gm. of fat and is receiving three daily doses of crystalline insulin, of 40, 15 and 40 units.

Urinalysis showed a trace of sugar. Microscopic examination showed that the epidermis was unchanged. There was some perivascular infiltration in the upper part of the corium; the collagen was moderately swollen, and the intercellular spaces were widened. A homogeneous eosin-staining area occupied almost all of the lower part of the corium and the subcutaneous tissue. This area was broken up somewhat by many small vessels, whose walls showed thickening with obliteration of the lumen and extensive perivascular infiltration.

A Case for Diagnosis (Poikiloderma Atrophicans Vasculare? Scleroderma?). Presented by DR. HOWARD MORROW, San Francosco (for the Division of Dermatology of the University of California Medical School).

E. B. L., a woman aged 54, a native of Central America, presents widespread areas of depigmentation, atrophy and telangiectasia associated with generalized pruritus, stomatitis and diarrhea. She previously was presented before the San

Francisco Dermatological Society on Dec. 3, 1937 (ARCH. DERMAT. & SYPH. **38**:140 [July] 1938) and before the joint meeting of the San Francisco and Los Angeles societies on Feb. 12, 1938 (ibid. **38**:504 [Sept.] 1938). Her condition remains unchanged.

DISCUSSION

DR. HOWARD FOX, New York: I was asked if I thought this condition was pinta. In my opinion it is not pinta, for several reasons. In the first place, there is no bluish pigmentation (I examined the patient from head to foot). Secondly, the Wassermann reaction was negative. In the third place, the patient came from Guatemala twenty years ago, and this eruption has been present for only twelve years. This leaves a period of eight years in this country prior to the development of the eruption, which excludes pinta. She certainly has a good deal of pigmentation. Whether the remainder of her skin, which is brownish, presents hyperpigmentation or her natural color I do not know. She has had, I understand, a good deal of ultraviolet irradiation.

DR. FRED WISE, New York: Two cases of a similar involvement have been demonstrated before dermatologic societies in New York under the diagnosis of anomalous pigmentary changes of unknown origin.

An almost exact replica of the clinical appearance of the eruption here presented may be found in the article by Habermann in J. Jadassohn's hand book (Handbuch der Haut- und Geschlechtskrankheiten, Berlin, Julius Springer, 1931, vol. 4, pt. 2, p. 859).

A Case for Diagnosis (Infiltrated Scaling Plaques of Legs, Thighs and Elbows, Followed by Atrophy). Presented by DR. H. E. MILLER, San Francisco (from the Division of Dermatology of the University of California Medical School).

H. C., a white woman aged 25, presents infiltrated symmetric reddish blue coalescing plaques topped by scales and crusts on the legs, posterior parts of the thighs, buttocks, knees and elbows. Some show superficial central ulceration. Between the areas of activity the skin shows telangiectasia and atrophy. The condition has been present for fifteen years. Microscopically, sections showed parakeratosis, thinning of the rete and almost complete obliteration of the basal layer. There was an increase in the number and dilitation of the vessels throughout the corium, with considerable diffuse chronic inflammatory reaction in the upper portions. This appeared in denser collections separated by bands of collagen fibers in the lower part of the corium. This patient was presented by Dr. Frances Torrey before the San Francisco Dermatological Society on April 22, 1938 (ARCH. DERMAT. & SYPH. **38**:1002 [Dec.] 1938). No appreciable change has occurred since then.

DISCUSSION

DR. FRED WISE, New York: The lesions overlying the shoulder and clavicles are analogous to those described by J. Csillag (in Neisser, A., and Jacobi, E.: Ikonographia dermatologica, Berlin, Urban & Schwarzenberg, 1909, vol. 4, p. 147) under the title dermatitis lichenoides chronica atrophicans. Lichen albus of Zumbusch probably belongs in the same category.

DR. OLIVER S. ORMSBY, Chicago: The only patients I have seen with lichen albus of the type just described presented no evidence of hypertrophic lesions, which in some areas in this case are pronounced. Taking it all together, hypertrophic lichen planus would seem to fit the picture best.

DR. H. E. MILLER, San Francisco: The diagnosis of hypertrophic lichen planus was considered seriously, but it was felt that the histologic picture ruled it out. Perhaps it would be wise to perform another biopsy.

A Case for Diagnosis (Dermatitis Repens? Acrodermatitis Continua of Hallopeau?). Presented by DR. C. J. LUNSFORD, Oakland, Calif.

F. E. M., a white woman aged 54, first seen on Feb. 1, 1938, states that she has had an eruption limited to the skin of her left thumb for about twenty-five

years. At first the lesions would clear under simple local therapy and recur only after several years. Later the flare-ups occurred every few months. The patient states that the skin would be perfectly normal in the meantime. Sometimes the lesions began as simple pustules and at other times as paronychia. She felt perfectly well otherwise.

Over the nail bed, tip and flexor surface of the left thumb was a red tense edematous inflammatory reaction covered by glazed, shiny epidermis. Over the thenar eminence were pea-sized deep-seated pustules with reddened bases. There was extreme tenderness.

Dr. A. P. Krueger, of the department of bacteriology at the University of California, after several noncontributory examinations obtained a positive culture from a pustule on the thumb. The organisms cultured were microaerophile type gamma streptococci and a nonhemolytic type of Staphylococcus aureus. Wassermann and Kline tests and urinalyses gave negative results. Repeated examinations both microscopic and cultural, for fungi gave negative results. Roentgenograms of the teeth showed multiple apical abscesses. The infected teeth were guardedly extracted and material from them sent to Dr. Krueger for culture. His findings included: type alpha hemolytic streptococci; microaerophile type alpha prime hemolytic streptococci and Staphylococcus aureus haemolyticus.

Local treatment has included wet compresses of sodium chloride, solution of boric acid and solution of mercury bichloride. Ammoniated mercury ointment also has been used at various times. Her present local treatment is with 15 per cent sulfur ointment.

Dr. Krueger prepared a lysate on the two occasions of the cultures. This has been applied locally without benefit. At the time the lysate was used the other drugs were withdrawn. I have been attempting to desensitize the patient by using an autogenous vaccine obtained from Dr. Krueger's culture of material from the skin. This later was combined with the vaccine made from the culture of material from the teeth. These vaccines have been given three times a week, the dose beginning with 0.01 cc. intradermally and gradually increasing as conditions permitted. She now is able to tolerate 0.1 cc. of the vaccine without a local or focal flare-up.

The patient's condition now is about as it was when I first saw her, four months ago. There have been flare-ups and remissions, apparently uninfluenced by treatment.

F. E. M. has just undergone a thorough general physical examination by Dr. Fletcher Taylor, Oakland, Calif. Infected tonsils were found.

Diagnoses considered include: (*a*) local bacterial infection of the skin; (*b*) pustular bacterid; (*c*) pustular psoriasis (no lesions of psoriasis elsewhere), and (*d*) fungous infection (no microscopic or cultural evidence).

DISCUSSION

Dr. George C. Andrews, New York: This patient presents a picture which I think is clinically acrodermatitis continua, because of the bacteriologic findings and the localization on one finger over such a long period. It does not seem to be characteristic of either pustular psoriasis of the extremities, as described by J. T. Ingram (*Lancet* **2**:13 [July 4] 1936) and H. W. Barber (Proc. Roy. Soc. Med. **26**:329 [Feb.] 1933), or of the pustular condition which G. F. Machacek and I have described as a bacterid which begins on the palms (Arch. Dermat. & Syph. **32**:837 [Dec.] 1935). I do think, however, that this woman's tonsils should be taken out, on the bare chance that the operation might influence the condition. I myself had erysipelas about eight or ten times over a period of years until eventually I had my tonsils out. I had a mild attack of erysipelas the day after my tonsillectomy. In the ten or twelve years since then I have had no further attacks. I believe many people who have abscessed teeth, as she has, also have infected tonsils.

Dr. George M. MacKee, New York: This eruption is difficult to diagnose. Acrodermatitis continua and dermatitis repens are progressive diseases—the lat-

ter usually, if not always, having an undermined edge. There are no distinct remissions and exacerbations. This eruption has no undermined edge; it is not definitely progressive, and there are remissions and exacerbations. According to the patient's statement, the skin is at times normal. The nail has exfoliated several times. Recurrence may begin as an inflammation near the nail or as a pustule in any area occupied by the previous eruption. Against the diagnosis of pustular bacterid and pustular psoriasis is the finding of pyogenic organisms in the pustules. I understand that repeated examinations for fungi have given negative results. The case does not look like one of malingering, but that possibility should be kept in mind.

DR. JOHN BUTLER, Minneapolis: I agree that dermatitis factitia should be considered.

DR. J. GARDNER HOPKINS, New York: I should like to ask the presenter whether the organisms were identified on stained smears. I gather from the history that several cultures were made.

DR. JOSEPH GRINDON, St. Louis: I questioned the patient in regard to the continuous character of the eruption. I received the impression that she had no intermissions but only remissions during the twenty-five years it has been present.

DR. C. J. LUNSFORD, Oakland, Calif.: In answer to Dr. Hopkins' question, the cultures were obtained with a great deal of difficulty.

Mycosis Fungoides. Presented by DR. G. V. KULCHAR, San Francisco (from the Department of Dermatology and Syphilology, service of Dr. Harry E. Alderson, Stanford University School of Medicine).

H. W., a white woman aged 47, presents a profuse generalized erythematosquamous eruption distributed in the form of well defined slightly elevated infiltrated plaques over the trunk, extremities and face. It has been present for nine years. She was presented at the joint meeting of the Los Angeles and San Francisco dermatologic societies on Feb. 12, 1938 (ARCH. DERMAT. & SYPH. **38**:497 [Sept.] 1938).

There has been no essential change since then.

DISCUSSION

DR. PAUL A. O'LEARY, Rochester, Minn.: I saw this patient for the first time five years ago. She then presented clinical and histologic evidence of mycosis fungoides. There were approximately six early fungating lesions, in addition to the typical plaques seen in this disease. She was given twelve treatments in the Kettering-Simpson hyperthermy unit in conjunction with the intramuscular use of ethylchaulmoograte. Over 500 cc. of ethylchaulmoograte was given, in doses of from 3 to 10 cc. Before coming under my care she had received considerable roentgen therapy on the West Coast. At that time there was extreme pruritus, while now there is little. Perhaps to many of the members the diagnosis of mycosis fungoides today would seem far-fetched. To me the therapeutic effect is striking. This case is the first in which I used ethylchaulmoograte and artificial fever, and from the result it would seem that a further trial of these agents is warranted.

Rhinoscleroma. Presented by DR. HARRY E. ALDERSON, San Francisco (from the Department of Dermatology of Stanford University School of Medicine).

B., a white woman aged 31, was presented before the San Francisco Dermatological Society on April 17, 1936 (ARCH. DERMAT. & SYPH. **36**:466 [Aug.] 1937). A more detailed report with photographs appeared in the ARCHIVES (**36**:1018 [Nov.] 1937). There is extensive involvement of the skin on the face, as well as in the more usual locations on the nose, hard palate and upper lip.

Rhinoscleroma. Presented by Dr. F. G. Novy Jr., Oakland, Calif. (from the Division of Dermatology of the University of California Medical School).

T. P. G., a Central American man aged 27, is presented because of involvement of the nares, hard and soft palate and alveolar margin, resulting in loss of the anterior upper teeth. A nodule is also present on the skin below the left inner canthus. At one time the picture was complicated by the presence of virulent diphtheria bacilli, without evidence of clinical diphtheria. The patient was presented before the San Francisco Dermatological Society on Dec. 4, 1936 (Arch. Dermat. & Syph. **36**:1241 [Dec.] 1937), and again by Dr. John M. Graves at

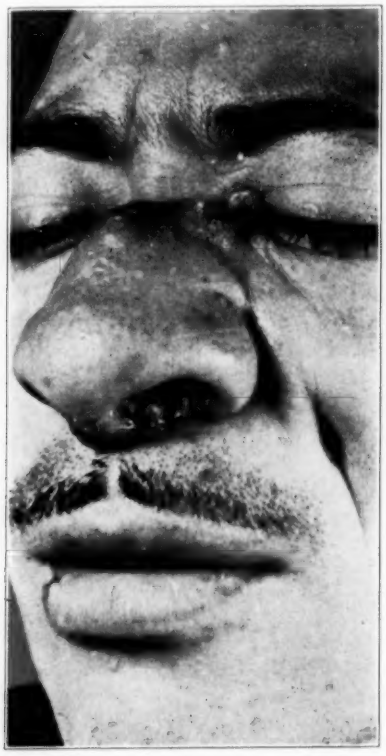

Fig. 1 (T. P. G.).—Rhinoscleroma.

the combined meeting of the Los Angeles and San Francisco societies on Feb. 12, 1938 (ibid. **38**:494 [Sept.] 1938).

There has been continued improvement since then, presumably because of roentgen treatment (fig. 1).

DISCUSSION OF CASES OF RHINOSCLEROMA

Dr. Charles C. Dennie, Kansas City, Mo.: I believe that the man, T. P. G., may have American leishmaniasis rather than rhinoscleroma, because of the extensive involvement of the upper jaw and other areas besides the nose. American leishmaniasis may be slow both in inception and in growth. There was a case in Kansas City, that of a Mexican, in which most of the face, including the upper and lower jaws and both eyes, was destroyed before death. I observed 2 or 3 cases at McGill University.

DR. GEORGE M. MACKEE, New York: I agree with the diagnosis of rhinoscleroma in Mrs. B., but the involvement is the most remarkable that I have ever seen. I have never known the disease to cover so much territory, especially so far from its original site in the nose and throat. Another remarkable feature is that it at first was limited to the usual site and later involved the face in the areas which had been the site of a severe contact dermatitis. It is unusual for rhinoscleroma to develop in previously irritated tissue as diseases such as lupus erythematosus and psoriasis so often do.

DR. HOWARD FOX, New York: I agree entirely with Dr. MacKee. I think every one here must consider this condition most unusual. I should like to ask the presenter whether in looking through the literature he has ever read of a similar involvement or has seen a photograph of rhinoscleroma of such extent on the cheeks.

DR. FRED D. WEIDMAN, Philadelphia: The histologic picture of the lesion of patient T. P. G. is not that of rhinoscleroma; the infiltration is almost purely of plasma cells, without any of the Mikulicz cells or Russell's fuchsin cells.

DR. HARRY E. ALDERSON, San Francisco: There is one weak point in the case of Mrs. B.; I have never been able to obtain cultures of Frisch bacilli, although I have tried many times. However, the bacilli showed up plainly in sections. Originally the nose presented enormous lesions and the cheeks were entirely free. The disease did not involve the cheeks until after the development of dermatitis venenata due to the application of nupercaine ointment by the roentgenologist during the administration of high voltage radiation directed at the palatal lesions. The rhinoscleromatous lesions then appeared and spread rapidly in exactly those areas involved by the dermatitis. The lesions on the eyelids are of recent origin. The palatal lesion has subsided about one half. The patient has had no roentgen treatment for a year.

Keratosis Follicularis (Darier). Presented by DR. N. N. EPSTEIN and DR. E. A. LEVIN, San Francisco.

A. S., a white woman aged 44, was first seen on June 4, 1937, because of an eruption involving the entire body. The condition is most severe in the groins and on the lower part of the abdomen, thighs and vulva. The lesions form diffuse hyperkeratotic, warty patches. In the most severe areas there are deep fissures bathed in a foul exudate. The patient's general condition is otherwise normal. Microscopic examination showed marked dyskeratosis and hyperkeratosis with considerable edema of the epidermis. There was a moderate lymphocytic infiltration beneath the epidermis. This patient was presented by Dr. C. E. Schoff, of Sacramento, Calif., before the American Dermatological Association in San Francisco in 1929, with a diagnosis of keratosis follicularis (ARCH. DERMAT. & SYPH. **21**:684 [April] 1930). She is shown again today because of the great progression of the process since that time.

Cutis Laxa, Abnormally Sensitive to Trauma. Presented by DR. DOUGLASS W. MONTGOMERY, San Francisco.

R. B., a white youth aged 18, complains of looseness of the skin and of extensive laceration in response to minor injury. Laxity of the skin, scarring and hyperextensibility of the joints of the fingers and toes are demonstrated. This patient was previously presented by Dr. J. D. Viecelli at the joint meeting of the Los Angeles and San Francisco dermatologic societies on Feb. 12, 1938 (ARCH. DERMAT. & SYPH. **38**:498 [Sept.] 1938).

DISCUSSION

DR. H. J. TEMPLETON, Oakland, Calif.: When this case was previously presented I suggested that it was an excellent instance of the Ehlers-Danlos syndrome. This includes hyperelasticity and friability of the skin, extensive scarring and hypermotility and hyperextensibility of the joints.

DR. OLIVER S. ORMSBY, Chicago: This case is of interest to me for the reason that in April Dr. W. W. Tobin and I presented a similar one before the Chicago Dermatological Society (ARCH. DERMAT. & SYPH. **38**:828 [Nov.] 1938), and in May a second was shown. Extreme examples are not infrequently seen in museums. Two types of dermatolysis or lax skin are recognized. The first occurs in Recklinghausen's disease, in which large masses of skin may depend from the face, neck, arms or other points. Pigmentation and tumors may or may not be present. The second variety, that shown today, consists of congenital abnormal laxation and friability of the skin, together with loose flexible joints, especially of the thumbs (Ehlers-Danlos syndrome). In our patient numerous scars had been induced by trauma insufficient to injure normal skin. The vulnerability of the skin resembles that seen in epidermolysis bullosa, in which a slight bruise causes injury.

Our histologic examination showed the blood vessels to be tortuous, which allowed them to be stretched without breaking. The collagen occurred in small long fibrils in place of the usual broad bundles, and the elastic fibers appeared wavy and were apparently longer than usual. These observations were early recorded by Unna and Williams.

DR. RICHARD S. WEISS, St. Louis: I have under observation at the present time a patient presenting all these signs, including the pseudotumors. The latter are evidently the results of injury, and changes in the elastic tissue result in balloon-like scarring. Such patients sometimes get extensive ulceration from mild injuries. The resulting scarring is thin and tissue paper–like. It is inclined to balloon up and produce a bulging tumor-like mass that feels almost cystic. In my patient the findings are all more exaggerated than those in the patient presented here. There are a great many more scars and pseudocysts.

DR. HOWARD FOX, New York: As Dr. Ormsby says, many persons with this extreme elasticity of the skin find their way into circuses. As a boy I remember seeing such a person. I also once saw a man who could actually pull his lower lip over his nose.

DR. JAMES H. MITCHELL, Chicago: In discussing the disease before the Chicago Dermatological Society I called attention to the fact that in a recent number of a popular weekly picture magazine a man was shown who was able to put six golf balls in his mouth, showing a remarkable extensibility of that orifice. I also called attention to the passage in the memoirs of Dr. George Henry Fox (Reminiscences, New York, Medical Life Press, 1926) in which he told of having difficulty in getting clinical material in the early days. He therefore borrowed material from the museum from time to time. One day he came in and, much to his surprise, his clinic was packed. He had borrowed a patient with cutis laxa for presentation and then discovered that the enterprising owner of the museum had been advertising this for weeks.

DR. JOSEPH GRINDON, St. Louis: This boy had defective dentition. A lateral incisor, I think it was, had not erupted. That is another feature characteristic of the Ehlers-Danlos syndrome.

Hereditary Hemorrhagic Telangiectasia. Presented by DR. A. E. INGELS (from Stanford University School of Medicine, Department of Dermatology, service of Dr. Harry E. Alderson).

J. M., an Austrian-Jewish furniture polisher aged 70, is presented because of widespread telangiectasia, involving the backs of the hands, arms, thighs, buttocks, legs, nose and cheeks. The mucous membranes are not involved.

Adherent subcutaneous masses were found in the buttocks at the first examination, and the Wassermann test was positive.

Since March 11, 1938, sodium bismuthate has been administered. The telangiectasia has not been altered by antisyphilitic treatment, but the gummatous lesions of the buttocks have continued to involute.

The patient was presented previously at the meeting of the San Francisco Dermatological Society on April 22, 1938 (ARCH. DERMAT. & SYPH. **38**:997 [Dec.] 1938).

Infra-red photographs of the forearms are shown in contrast with ordinary photographs.

DISCUSSION

DR. RICHARD S. WEISS, St. Louis: I do not think that this patient has Osler's hereditary telangiectasia. In Osler's disease there are telangiectatic lesions in the nose and mouth, and bleeding from these areas is frequent. My diagnosis would be a form of telangiectatic nevus. Nevi have a tendency to be hereditary also. If that is not the diagnosis, then I believe the condition belongs with the idiopathic atrophies with an unusual amount of telangiectasia.

DR. RUBEN NOMLAND, Iowa City: I agree with Dr. Weiss that this picture is not Osler's disease as it is ordinarily considered. I believe, however, that the man does present a syndrome that has been described by Dr. S. W. Becker, of Chicago, under the name generalized acquired telangiectasia (ARCH. DERMAT. & SYPH. **14**:387 [Oct.] 1926).

Extracellular Cholesterosis of Urbach. Presented by DR. KENDAL FROST and DR. C. R. ANDERSON, Los Angeles.

This patient, a Mexican laborer aged 35, first noted about Nov. 15, 1937, a soreness and tenderness, aggravated by motion, in the knees and popliteal spaces and on the ankles, feet, shoulders, elbows, wrists and fingers. In December he noted the development of small round yellowish brown elevated papules on the palmar surfaces of the fingers. These lesions became confluent, and the palmar surfaces became so thick and tender that the patient had to stop work. Within the next month or two similar lesions appeared over the backs of the hands and fingers, on both elbows and on the inferior surface of each buttock. Smaller isolated lesions appeared on the forearms, the left temple and the right side of the neck. The individual papules tended to increase in size and coalesce, forming yellowish brown plaques. The epidermis over these areas was thin, atrophic and crinkled and showed a superficial delicate scaling. There was a tendency to central involution, leaving central depressed areas. On diascopic pressure the infiltrated lesions showed a distinct underlying yellow color. In the center of some of the papules hemorrhagic necrosis occurred. These lesions healed, leaving depressed atrophic scars. Small hemorrhages varying in size from that of a pinpoint to that of a pinhead also occurred in some areas, especially about the nail folds and finger tips.

The past and family history were unimportant. Physical examination showed nothing abnormal but the cutaneous lesions and associated pain and stiffness of the joints. There were no mucosal lesions, and neither spleen nor liver was enlarged. The patient was hospitalized at the Santa Fe Coast Lines Hospital on December 13, for a tonsillectomy. From January to March 1938 he received forty intragluteal injections for arthritis. He was hospitalized again on March 11, and a tentative diagnosis of leprosy was made. I first saw him on March 18 and made a tentative diagnosis of xanthoma tuberosum. At that time involution had already begun in some of the larger plaques and the infiltration of the palms was somewhat less.

During hospitalization Wassermann, Kahn and Klein tests were normal. A blood count and urinalysis showed no abnormality. Scrapings from nodules and pharyngeal and nasal smears showed no leprosy bacilli. A dextrose tolerance test gave a normal result. The uric acid content of the blood was 2.6 mg. per hundred cubic centimeters. The basal metabolic rate was +22 per cent. The blood cholesterol content was 80 mg. per hundred cubic centimeters on March 22, and on March 25, 102.5 mg.; control blood showed 133.3 mg. per hundred cubic centimeters. Roentgen studies were made by Dr. D. R. MacColl. Those of the chest

showed slight pleural thickening over the apexes of both lungs and a moderate amount of hilar scarring but no evidence of active disease. Stereoscopic roentgenograms of the skull were normal. Films of both hands showed only some thinning of the cartilages of all the interphalangeal joints and some demineralization of all the bones—probably an atrophy of disuse.

Microscopic examination of sections of a lesion from the right elbow showed relative and absolute hyperkeratosis of the epidermis. Portions of the epidermis appeared normal, but other portions showed vacuolation of the cells of the rete and stratum basale. A decided cellular infiltration between the collagen fibers extended from the papillary bodies down into the lower portions of the cutis. In the central portions of the lesion the infiltrate was almost solid. Along the borders it was arranged in a perivascular manner. It consisted of proliferated mesenchymal cells of the perivascular spaces, mononuclear cells, fibroblasts, polymorphonuclear leukocytes, a few eosinophils and nuclear and cellular débris. In some of the areas more severely involved there were degenerative changes in the connective tissue fibers, with rarefaction, fragmentation and tinctorial changes. In various parts of the section there were vascular dilatation, endothelial swelling and proliferation, perivascular fibrosis and narrowing of the lumens of the vessels. Nowhere was there evidence of so-called xanthoma cells.

Sudan III stain showed small brownish red droplets heavily scattered throughout the section. These for the most part were extracellularly disposed, although some of the fat droplets were to be seen in the capillary walls and occasionally apparently also in some of the connective tissue cells.

NOTE.—Subsequently the active lesions on the skin were treated with fractional doses of roentgen rays, resulting in fairly rapid absorption. A few months later the man died. At autopsy none of the fat deposits were demonstrable in any of the tissues. There was a retroperitoneal lymphosarcoma, with involvement of the intestines, and multiple perforations of the small intestine.

DISCUSSION

DR. FRED D. WEIDMAN, Philadelphia: Clinically the individual lesions are compatible with extracellular cholesterosis, and I accept that diagnosis. The distribution preeminently over the shins does not appear in this patient, however. By now the frozen sections have faded. I understand, though, that at one time they did exhibit the material in proper position.

DR. KENDAL FROST, Los Angeles: Until further studies are made it would probably be better to call the disease lipoidosis. It seems to correspond to C. W. Laymon's description of cholesterosis (ARCH. DERMAT. & SYPH. **35**:269 [Feb.] 1937), however.

Hydroa Vacciniforme. Presented by DR. MERLIN TREVOR-ROPER MAYNARD, San Jose, Calif. (from the Department of Dermatology of Stanford University School of Medicine, service of Dr. Harry W. Alderson).

A. S., a white boy aged 15 years, was first seen on April 26, 1937. Three days after he was vaccinated, at the age of 4 years, an eruption appeared. This has recurred frequently since then, usually following exposure to sunlight, cold and wind.

When I first saw him the face, ears and backs of the hands were covered with purulent bloody crusts. Considerable secondary infection was obviously present. This was easily controlled with dressings of ammoniated mercury ointment. The eruption has cleared entirely at different times when he has stayed indoors all day long. Exposure outdoors has resulted in recurrences, however. The use of an ointment containing quinine and one containing dihydroxyquinoline sulfate seemed to afford some protection. Treatment other than this, including roentgen irradiation, exposures to ultraviolet rays, the use of autogenous vaccine, elimination diet and

administration of sodium gold thiosulfate and of capsules of pancreatin, was of no apparent help. On May 20, 1937, he was tested with a series of light filters, borrowed from Dr. H. J. Templeton, of Oakland, Calif., to determine reactions to light of various wavelengths. His back was exposed through these filters for one-half hour in intense sunlight on the top of an office building. Twenty-four hours later none of the areas had reacted except the area where no filter was used, and this reacted with normal sunburn. This suggests that the sensitivity was more or less local.

Urinalysis showed no porphyrin. A blood count was normal except that the differential count showed 11 per cent monocytes.

DISCUSSION

Dr. Donald M. Pillsbury, Philadelphia: I believe it was recorded in the protocol that this boy showed no porphyrin in the urine. I should like to point out that apparently in many cases the amount of porphyrin in the gastrointestinal tract may be of more importance. Erich Urbach has recently published an article describing a number of cases of light-sensitive dermatoses in which there were bacteriologic disturbances of the gastrointestinal tract (*Klin. Wchnschr.* **17**:304 [Feb. 26] 1938). These included a suppression of the colon group and the presence of large numbers of streptococci. Some of the patients responded to the feeding of colon bacilli. Dr. Stokes has observed a case of similar involvement recently. Porphyrin is normally present in the gastrointestinal tract—I believe in the form of coproporphyrin. In this disease, however, there may be a marked increase in this and other forms of porphyrin. Apparently this may be caused by organisms which are capable of synthesizing these substances in the gastrointestinal tract.

Dr. Paul A. O'Leary, Rochester, Minn.: The Mayo Clinic's efforts to evaluate the significance of porphyrins in the various dermatoses have been rather disappointing, especially in patients with light sensitivity. Uroporphyrin is apparently a unique and rare substance which is difficult to obtain. My associates and I have been working with coproporphyrin, which is demonstrable in the feces and urine. In practically none of the patients with light sensitivity were we able to demonstrate coproporphyrin to any extent; in fact, with many other dermatoses coproporphyrin is present in greater amounts. Accordingly it would appear that the porphyrins are not the etiologic agents in light sensitivity, as they have been thought to be for several years.

Carcinoma of Fauces. Presented by Dr. A. E. Ingels, San Francisco (from the Stanford University School of Medicine, Department of Dermatology, service of Dr. Harry E. Alderson).

J. J. P., a white laborer aged 47, was first seen May 19, 1938, because of a swelling of his neck noted a week before. He stated that he smoked one-half sack of tobacco and drank 5 or 6 ounces (150 to 180 cc.) of liquor daily. He had had gonorrhea in 1915 and again in 1921; it cleared without residual symptoms. He had sexual intercourse two months ago but has noted no penile lesion, cutaneous rash or falling of the hair. The swelling of his neck was accompanied by cough, sore throat and dysphagia. He was seen elsewhere, and a dark field examination of material from his throat was reported as showing spirochetes. The Wassermann reaction of his blood was negative. An injection of neoarsphenamine was given.

When I first saw him, his tongue was coated and the mucosa of the mouth generally inflamed. The tonsils, soft palate, uvula and fauces were occupied by ulcerated fungating masses which were firm to palpation. The submaxillary glands were swollen. His hands presented a dermatitis suggesting pellagra. A generalized maculopapular eruption suggesting a "toxic" origin was present. Both of these manifestations have now largely cleared. There is no history of ingestion of drugs other than neoarsphenamine. Dark field examination showed only

spirilla of Vincent. The Wassermann reaction has been repeatedly negative. Cultures of material from the throat showed a predominating growth of Streptococcus viridans. Smears showed many fusiform bacilli and spirilla.

Microscopic examination showed the growth to be squamous cell carcinoma. Roentgen treatment has been started.

DISCUSSION

DR. BEDFORD SHELMIRE JR., Dallas, Texas: I believe the dermatitis on the hands is typical of pellagra. The man is probably not eating properly because of the extensive lesion in the throat.

A Case for Diagnosis (Darier's Disease?). Presented by DR. ERVIN EPSTEIN, Oakland, Calif.

L. B., a white boy aged 16, is presented because of translucent firm hyperkeratotic papules on the forehead, cheeks, elbows and knees. He was presented before the San Francisco Dermatological Society on April 22, 1938 (ARCH. DERMAT. & SYPH. **38**:998 [Dec.] 1938).

Since then the circular outlines of the patches have disappeared, owing to the development of new lesions in the hitherto clear centers, and new lesions have appeared on the arms and thighs. He has received two blood transfusions, and the lesions now appear much redder than before. Recently several pustular lesions have appeared. In addition to the laboratory work previously reported, intradermal tests with 1:1,000 dilutions of human and of bovine tuberculin gave negative results. Chemical studies of the blood gave the following results:

Nonprotein nitrogen	120.0	mg. per 100 cc.
Creatine	2.2	mg. per 100 cc.
Serum albumin	5.01	mg. per 100 cc.
Serum globulin	2.0	mg. per 100 cc.
Carbon dioxide–combining power	29	vol. per cent
Chlorides	555.8	mg. per 100 cc.
Calcium	6.5	mg. per 100 cc.
Phosphorus	7.27	mg. per 100 cc.
Phosphatase	16	units

The Kolmer and the Kahn reaction of the blood were negative. Roentgenograms of the long bones showed renal rickets.

Microscopic sections stained with hematoxylin and eosin are presented. Stains for amyloid gave negative results.

DISCUSSION

DR. HOWARD FOX, New York: Was Darier's disease ruled out histologically?

DR. JAMES H. MITCHELL, Chicago: The diagnoses that were discussed at the meeting of the San Francisco Dermatological Society included Darier's disease, avitaminosis, xeroderma pigmentosum and tuberculid.

DR. GEORGE M. MACKEE, New York: The clinical diagnosis is Darier's disease. I could not identify the disease histologically, however, from the sections I saw.

DR. H. N. COLE, Cleveland: This peculiar eruption reminds me somewhat of that described by C. Frazier and Ch'Uan-K'Uei Hu (*Arch. Int. Med.* **48**:507 [Sept.] 1931), associated with a lack of vitamin A. The boy also gave a history of not eating well.

DR. FRED D. WEIDMAN, Philadelphia: I agree with Dr. MacKee that a keratinous plug can be seen in most of the lesions on the forehead. However, in the histologic section there is nothing that conforms to the picture of keratosis follicularis. I suspect that the sections are not representative of the clinical lesions and that if another biopsy specimen were taken it would show the histologic picture of Darier's disease.

DR. PAUL A. O'LEARY, Rochester, Minn.: My thoughts in regard to this patient are different from those expressed so far. I believe this boy has disseminated xanthomatosis.

DR. HOWARD FOX, New York: I have not seen Dr. Frazier's patients with avitaminosis, but I have seen a number of examples of a similar condition in the tropics, especially in Yucatan. The pediatricians in that country are familiar with the eruption. It looks more or less like ordinary keratosis pilaris.

A Case for Diagnosis (Generalized Macular Eruption). Presented by DR. N. N. EPSTEIN and DR. E. A. LEVIN, San Francisco.

B. S., a youth aged 17, was first seen on March 29, 1938, because of a generalized eruption extending from the neck to the feet. The lesions consist of numerous macular areas about 1 to 1.5 cm. in diameter, some slightly redder than others. Several show a slight scale. They are asymptomatic. The lesions do not swell on friction. A blood count and urinalysis were normal. The Wassermann and the Kahn test were negative. After a moderately severe generalized sunburn, the eruption apparently disappeared, but has recurred since. On clearing, many lesions leave depigmentation and apparent atrophy.

Microscopic study showed parakeratosis, with some edema of the epidermis and a moderately dense infiltrate of lymphocytes in the corium. There was considerable increase in pigment. Sections stained to demonstrate mast cells showed none.

DISCUSSION

DR. OLIVER S. ORMSBY, Chicago: The most likely diagnosis is parapsoriasis. The distribution of a macular slightly scaling disease with no subjective sensations and comparatively long duration are all favorable to this diagnosis. Occasionally the picture resembles maculopapular syphiloderm clinically, but in this instance the long duration and lack of concomitant symptoms rule out syphilis.

DR. CLARK W. FINNERUD, Chicago: I came to the conclusion that the condition must be an unusual example of parapsoriasis. Pityriasis rosea is suggested by the configuration of the lesions and a few macular lesions that showed central clearing. I understand that syphilis has definitely been ruled out. Recently I have observed a case of phenolphthalein eruption which closely resembled that seen here. The largest lesions—and they were numerous—were the size of finger nails. The disease had been present six or eight months. The patient gave a history of having begun to take "ex-lax" shortly before its onset. Although I did not question him, I wondered if dermatitis medicamentosa had been considered.

DR. N. N. EPSTEIN, San Francisco: We obtained no history of ingestion of drugs.

Lymphoblastoma (Leukaemia Cutis?). Presented by DR. C. J. LUNSFORD, Oakland, Calif.

H. K., a white man aged 61, was referred by Dr. Leland H. Taylor, Oakland, Calif.

When the patient was a child of 6 or 7 years, chorea developed. About thirty-five years ago, for two or three years he had a vesicular eczema ("salt rheum") of the webs and sides of his fingers. This disappeared without any particular treatment. About thirty years ago a persistent erythema developed on the upper and inner parts of the thighs. There was no scaling and no symptoms. About twenty-five years ago several Wassermann tests of the blood were made because of nervousness and various phobias. The results were negative. In spite of this he received three injections of arsphenamine and two of neoarsphenamine and three years of subcutaneous injections every other day of a drug the name of which he does not know.

It was during the year following these injections that dryness and scaling, but no erythema, began to develop on the ankles and the outer aspects of the legs.

This spread slowly and did not itch. About ten years ago it developed on the thighs and continued to spread until it included the entire body except the face and hands.

Shortly after this infiltrated papules began to develop on various parts of the skin. These have gone on to central necrosis and eventual involution, leaving depigmented atrophic scars. New ones have continued to develop. About a year ago erythema first appeared about the ankles and rapidly spread to other parts of the body. It has become much worse during the last three months. During this time there have been varying degrees of itching.

About two months ago pain developed in the right shoulder, radiating from the base of the neck down the right arm. This was associated with numbness and stiffness of the right thumb and index and middle fingers, which have forced him to discontinue his work as a box and crate maker for the first time.

For years the patient's mouth has been periodically sore and inflamed. Small indolent ulcers with yellowish necrotic centers have appeared in various locations on the buccal and lingual mucosa.

At present the skin presents a generalized dry infiltrated lichenified exfoliating erythroderma. Superimposed on this are multiple pea-sized papules, some of which show beginning necrosis. In others the necrosis is more advanced and may be expressed as a firm black central plug. There are also innumerable white atrophic scars. In the mouth are several small reddened papules and ulcers. There is generalized lymphadenopathy, dominant in the inguinal region, where some of the glands are the size of a walnut. The liver and spleen are not palpable.

Microscopic examination of sections of skin and of a lymph node was reported by Dr. Paul Michael, of Oakland, Calif., as showing some acanthosis of the epidermis. Throughout the corium were closely packed large clear cells, oval and round and intermixed with old blood pigment. The lymph node showed a thickened capsule with a fairly well preserved architecture. The sinuses were engorged with fresh and old blood pigment, and the capillaries traversing the node were distended with blood. There was an intense reticulum cell hyperplasia. The lymph follicles were numerous, and the germinal centers showed hemorrhagic infiltration. Dr. Michael concluded that the microscopic structure of the skin and the lymph node were compatible with a diagnosis of leukemia.

A sternal puncture was done by Dr. Michael, who reported the differential count on the bone marrow to show: neutrophil leukocytes 15 per cent, eosinophil leukocytes 3 per cent, basophil leukocytes 1 per cent, stab nuclears 15 per cent, metamyelocytes 3 per cent, myelocytes 10 per cent, myeloblasts 2 per cent, monocytes 3 per cent, lymphocytes 36 per cent and lymphoblasts 12 per cent. He felt that this bone marrow picture was not in keeping with myelogenous leukemia but could fit in with a lymphatic type of leukemia.

On April 24, 1938, a blood count showed 5,000,000 erythrocytes and a hemoglobin concentration of 90 per cent. There were 38,200 leukocytes. A differential count showed: 76 per cent polymorphonuclears, 19 per cent small lymphocytes, 2 per cent large lymphocytes, 2 per cent immature cells and 1 per cent eosinophils. On April 28 there were 32,050 leukocytes, and a differential count showed 80 per cent polymorphonuclears, 15 per cent small lymphocytes, 2.5 per cent large lymphocytes and 2.5 per cent eosinophils. On May 27 there were 20,100 leukocytes, and the differential count was 73 per cent polymorphonuclears, 19 per cent small lymphocytes, 1.5 per cent large lymphocytes and 6.5 per cent eosinophils. On June 4 there were 19,800 leukocytes, of which 76 per cent were polymorphonuclears, 22 per cent small lymphocytes, 1 per cent large lymphocytes and 1 per cent eosinophils.

Urinalysis and the Wassermann test gave negative results.

Between May 10 and June 1 the patient has had a total of ⅜ skin unit of roentgen rays from the waist up, and ¼ skin unit to the areas below the waist. He has used olive oil locally.

Recently he has begun to feel weak and listless.

Coccidioidal Granuloma. Presented by DR. H. E. MILLER, San Francisco (from the Division of Dermatology of the University of California Medical School).

A. J. R., an Italian man aged 40, presents an unusual picture of coccidioidal granuloma characterized by brawny induration and erythema of the nose, upper lip and cheeks. He was presented before the San Francisco Dermatological Society on Dec. 4, 1936 (ARCH. DERMAT. & SYPH. **36**:1240 [Dec.] 1937) and again at the joint meeting of the Los Angeles and San Francisco societies on Feb. 12, 1938 (ibid. **38**:499 [Sept.] 1938).

There has been no notable change since then (fig. 2).

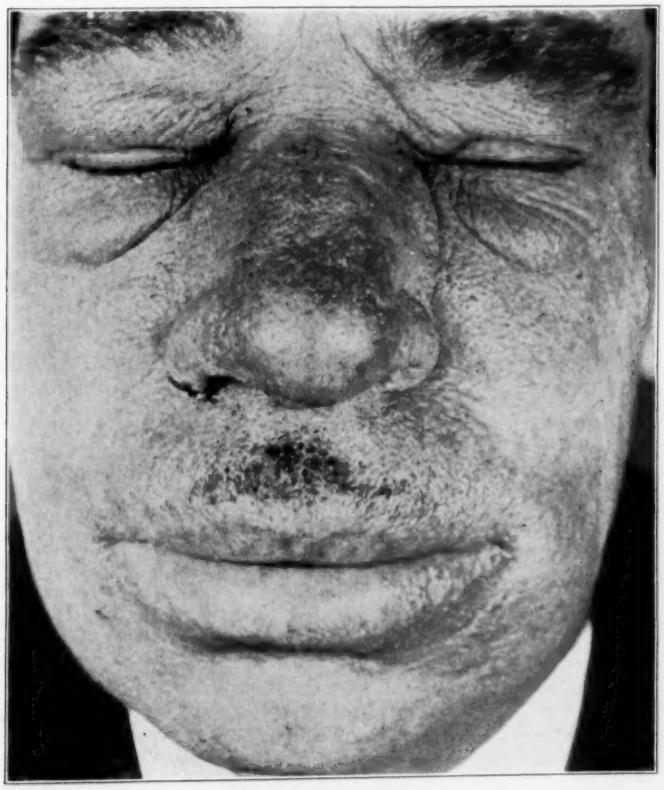

Fig. 2 (A. J. R.).—Coccidioidal granuloma, showing edema of the nose and midportion of the face, with granulomatous lesions in the nostril.

Coccidioidal Granuloma. Presented by DR. H. E. MILLER, San Francisco (from the Division of Dermatology of the University of California Medical School.)

H. S. H., a white ranch worker aged 43, was first seen on Dec. 28, 1937. One month before entry he contracted a "cold" with rhinitis, sore throat, cough and expectoration. About this time he noticed a swelling on his lower lip.

Examination showed a granulomatous mass involving most of the lower lip, most evident on the right side. Numerous superficial small abscesses exuded pus and serum. The lip was not particularly painful. The lymph nodes in the right submaxillary region, the axillas and the epitrochlear and inguinal regions were enlarged. The physical examination showed no essential abnormality except a blood pressure of 184 systolic and 110 diastolic.

A blood count showed a hemoglobin concentration of 106 per cent. There were 5,220,000 erythrocytes and 8,800 leukocytes. A differential count showed 70 per cent polymorphonuclear neutrophils, 26 per cent lymphocytes, 3 per cent monocytes and 1 per cent eosinophils.

A smear of pus in 15 per cent sodium hydroxide revealed two round double-contoured bodies suggestive of Coccidioides immitis.

Microscopic examination of sections showed chronic granuloma. No coccidioides organisms were seen.

A cutaneous test with coccidioidin gave negative results.

Roentgenograms of the chest showed only an increase in the hilar shadows.

Kolmer and Kahn tests were negative.

Cultures of material from the lip showed a fluffy white mold with septate mycelium.

Inoculation of a guinea pig showed Coccidioides immitis.

Roentgen therapy was begun on Jan. 5, 1938. A total of 500 roentgens unfiltered, divided into five weekly doses, produced great improvement of the local lesion (fig. 3).

Fig. 3 (H. S. H.).—Coccidioidal granuloma, showing solitary ulcerated nodule.

DISCUSSION

DR. CHARLES C. TOMLINSON, Omaha: I have observed but 3 cases of coccidioidal granuloma, 2 of which Dr. Paul Bancroft and I have reported as instances of cure following the intravenous use of antimony and potassium tartrate and roentgen irradiation to the lesions (*J. A. M. A.* **91**:947 [Sept. 29] 1928). Both of the patients have remained well. This experience makes me wonder why our results were so good or why others have not had similar experience.

DR. W. H. GUY, Pittsburgh: My entire series consists of 1 case. It is the first case in which antimony and potassium tartrate and roengen rays were used. The patient was a man with an extensive involvement. He is well today.

DR. RICHARD S. WEISS, St. Louis: I have observed only 1 case of coccidioidal infection in St. Louis. The patient had no cutaneous and no pulmonary lesions, but he did have arthritis, and the organism was cultured in material from the affected joints. He acquired his disease in California, and he died in St. Louis.

DR. KENDAL FROST, Los Angeles: Recently when I was in Bakersfield, which is about the center of the area in which the disease is prevalent, I saw several patients with coccidioidal granuloma in the office of one of the local physicians. He said that almost every practitioner in the community had a number of cases on his regular list. So it is a common disease in that location.

DR. HARRY E. ALDERSON, San Francisco: A patient in the Stanford University Hospitals now has typical coccidioidal pulmonary lesions, from which organisms have been recovered. She had erythema nodosum, often seen with this condition. She had never been in or near the San Joaquin valley, but just before her infection started she had helped to clean the outside and the inside of a dirty trailer that had been traveling through that section.

DR. FRED D. WEIDMAN, Philadelphia: May I ask whether the particular dermatologic expression of patient A. J. R. is unique for coccidioidal granuloma? The lesions I saw on my last visit to the Pacific coast looked more like scrofuloderma. Those illustrated in the literature are granulomatous, making one think of tuberculosis verrucosa cutis. This lesion looks more like leukaemia cutis.

DR. WILLIAM H. GOECKERMAN, Los Angeles: Apropos of Dr. Weidman's remarks, I am often struck by the versatility of this organism in producing clinical pictures.

DR. H. E. MILLER, San Francisco: These patients, especially A. J. R., present unusual clinical pictures for coccidioidal granuloma. I presented them simply for that reason.

It is always of interest to hear reports of 1 or 2 cures of coccidioidal granuloma with different drugs. When one tries to repeat the therapeutic procedure in another case one generally is not successful. Over fifty drugs have been used in the treatment of coccidioidal granuloma, and with some fifteen of them cures have been reported. In evaluating treatment it must be realized that probably in a large number of cases the disease clears spontaneously.

Blastomycosis. Presented by DR. E. A. LEVIN, San Francisco (from the Division of Dermatology of the University of California Medical School).

J. R., a white man aged 40, presents severe generalized blastomycosis. Symptoms first pointed to involvement of the urinary and the respiratory tract. Progressive loss of weight, pains in the joints, abdominal distress and most recently granulomatous subcutaneous abscesses followed. He was presented at the joint meeting of the Los Angeles and San Francisco dermatologic societies by Dr. F. G. Novy Jr. on Feb. 12, 1938 (ARCH. DERMAT. & SYPH. **38**:500 [Sept.] 1938).

He was subsequently treated with roentgen rays. An area on the dorsum of the right hand received the largest total amount. This was 1,250 roentgens and was given in fractional doses of 75 r once a week, filtered with 0.25 mm. of copper and 1 mm. of aluminum.

From January 4 to March 21 he was given 50 per cent thymol in oil orally three times a day, the dose beginning with 5 and increasing to 12 drops. During this time a new lesion about 1.5 cm. in diameter developed on the dorsum of the right forearm.

On March 21 he was given ¼ teaspoonful (about 1 cc.) of a saturated solution of sodium iodide three times a day. The dose was rapidly increased to 1 teaspoonful (about 4 cc.) three times a day. This was continued until April 18, when the patient complained of severe pain in the right shoulder and epigastric distress. Physical examination revealed no cause for this pain. The cutaneous lesions continued to progress.

On May 17 sulfanilamide medication was begun but was discontinued after two days because of severe nausea and vomiting.

Potassium iodide therapy was begun on May 24. There has been some improvement in the lesion on the right hand in the last week.

DISCUSSION

DR. OLIVER S. ORMSBY, Chicago: Seeing typical blastomycosis in California is of historical interest to me. Many years ago, when my associates and I were studying this disease in Chicago, Dr. Morrow and others were studying granuloma coccidioidale in California. It appeared for some time that the latter disease was restricted to California and that blastomycosis did not occur here. Time has shown that blastomycosis has a wide distribution throughout the United States, with an

occasional case on the western coast, but coccidioidal granuloma is still largely a California disease with an occasional case elsewhere. It was thought early that blastomyocisis was a purely cutaneous disease, but in 1903 with Dr. H. M. Miller I presented a case of generalized involvement (*J. Cutan. Dis.* **21**:121, 1903), and numerous others followed. On the other hand, the coccidioidal disease was early recognized as frequently systemic, the cutaneous involvement often being of minor importance. In the major portion of cases of blastomycosis the clinical picture is uniform and characteristic, and a positive diagnosis can usually be made. In the coccidioidal disease the cutaneous lesions are multiform, sometimes resembling other conditions, and the diagnosis often has to be made on the basis of the microscopic picture. It may be concluded that as a rule blastomycosis is predominantly a cutaneous disease with occasional systemic invasion, and coccidioidal disease is more often systemic with comparatively minor cutaneous involvement.

Dermatomyositis. Presented by DR. H. J. TEMPLETON, Oakland, Calif.

J. A., a Dane aged 54, is presented because of a pruritic erythematous scaly eruption associated with pains and stiffness in his muscles and joints. He was presented before the San Francisco Dermatological Society on April 22, 1938 (ARCH. DERMAT. & SYPH. **38**:1001 [Dec.] 1938).

Subsequently a further medical study was made by Dr. Fletcher B. Taylor, Oakland, Calif. Mild chronic tonsillitis was noted. Dental roentgenograms, an electrocardiogram and a determination of the basal metabolic rate showed no abnormality. Roentgenograms of the right hand showed some osteoporosis of the phalanges.

Treatment has consisted of the administration of 1 grain (0.06 Gm.) of thyroid daily, of yeast by mouth and of soothing topical applications.

At present he shows only slight erythema and puffiness of the face and residual slightly scaly dermatitis of the radial aspect of the forearms, elbows, palms and dorsa of the hands. Marked pigmentation, largely from sun bathing, is present. Tenderness and deep induration of the hands and wrists still can be noted. He cannot fully extend his fingers. There is no evidence of scleroderma.

DISCUSSION

DR. PAUL A. O'LEARY, Rochester, Minn.: This patient presented a moderately advanced stage of dermatomyositis and demonstrated clearly the atrophy of the shoulder girdle group of muscles, which is a prominent feature of this disease. I have recently seen this disease in a number of children as well as adults and have used a great variety of therapeutic measures with different degrees of success. I have been impressed with the frequency with which it may undergo spontaneous arrest. The atrophy and disability remain, while the progressive muscular involvement ceases. On the other hand, chiefly in children, this disease is frequently fatal. At the moment my associates and I are treating these patients with oxygen, typhoid vaccine and aminoacetic acid. Vaccine therapy and the various nonspecific measures have definite merit, but the intensive use of oxygen, either in an oxygen tent or by mask, results in considerable symptomatic relief.

DR. W. H. GUY, Pittsburgh: I have under observation at the present time a case of unusually severe dermatomyositis. There are an intermittent septic type of temperature curve, attacks of dyspnea and tachycardia, progressive muscular atrophy and loss of weight. Badly infected teeth were found. These were removed, and a green streptococcus was cultured. During acute febrile attacks it has been possible to recover a green streptococcus from the blood stream but not during the interims between attacks. These findings may be of significance. As. Dr. O'Leary has indicated, it would seem that recovery when it has occurred has been spontaneous. Aminoacetic acid has been suggested therapeutically, and I have been feeding the patient large amounts of gelatin with that in mind. It might be interesting in Dr. Templeton's case to examine sections of muscle microscopically for perivascular infiltration. In more advanced sections muscular degeneration becomes evident microscopically. With the amount of degeneration of muscle fiber that has occurred, a relatively high creatinine concentration in the urine would likely be found.

A Case for Diagnosis (Panniculitis?). Presented by Dr. Harry E. Alderson, San Francisco (from the Stanford University School of Medicine, Department of Dermatology and Syphilology).

L. P., an Italian woman aged 38, is presented for consideration of numerous well circumscribed firm, slightly pigmented and irregularly nodular masses adherent to the skin and involving the subcutaneous tissue. These have been developing during the past eight months on the posterior aspect of the right arm, both thighs and the buttocks.

She was presented at the joint meeting of the Los Angeles and San Francisco dermatologic societies on Feb. 12, 1938 (Arch. Dermat. & Syph. **38**:493 [Sept.] 1938).

Miscroscopic study reported by Dr. A. E. Ingels showed dense bands of fibrous tissue throughout the subcutaneous tissue, with areas of lymphocytic infiltration and multinucleated giant cells of the foreign body type. Numerous areas showed disappearance of subcutaneous fat, leaving lacunas. Stains for fat showed little or none in these areas. There was no tuberculous or sarcoid structure; there were no foam cells and no xanthoma cells. Stains for acid-fast bacilli failed to disclose tubercle bacilli, and no other organisms were demonstrable. The microscopic diagnosis was chronic panniculitis.

DISCUSSION

Dr. Richard S. Weiss, St. Louis: In my opinion this patient presents typical camphor liniment tumors of the type so well described some years ago by W. H. Mook and W. G. Wander (*J. A. M. A.* **73**:1340 [Nov. 1] 1919). The lesions which they described were nodules and definitely extended along lymph channels. The sections presented today did not show the typical granulomatous reactions that are associated with foreign body tumors, although in some areas there were traces of this. The patient told a clear story of a serious operation in 1923. She was disabled for a long time, and in her own words she "had got a great many needles stuck into her arms and thighs." I have been told that the physician who operated on the patient stated that no camphor liniment had been injected. However, it is possible that his recollection is faulty, as the operation was done many years ago.

Dr. Clark W. Finnerud, Chicago: I agree with what Dr. Weiss has said, aside from the fact that in the sections that I looked at there was a fine foreign body reaction, a chronic granuloma with plenty of giant cells involved. I think that the lesion is a foreign body tumor, the result of injections of camphor liniment.

Dr. Oliver S. Ormsby, Chicago: Clinically, it is a classic tumor due to camphor liniment or liquid petrolatum. At about the time she was operated on the former drug was on the market for use by injection. After producing trouble for a number of years, it was withdrawn.

Dr. Harry E. Alderson, San Francisco: Even if the condition were due to injections of camphor liniment or another substance, it is still panniculitis. Answering the comments about camphor liniment, I should say that it would be extremely unusual for injections to be given near the popliteal region and in the other places where these lesions appear.

Dr. C. Guy Lane, Boston: At the Massachusetts General Hospital there have been 3 cases of possible Weber-Christian type of panniculitis, in 2 of which the diagnosis has been proved conclusively. The patients were in their teens. Indurated areas with some slight tenderness developed spontaneously on the legs and gradually involuted. They left round superficial crater-like depressions, which gradually became soft and smooth. No indurated areas like those seen here remained.

Dr. Richard S. Weiss, St. Louis: Dr. Alderson stated that injections of camphor liniment would not be given near the popliteal spaces. I agree with that. However, characteristically tumors caused by liquid petrolatum can and often do arise at a distance from the point of injection. The oil seems to travel down the lymphatic spaces, and in the course of months or years the tumors appear at distances of 3 to 18 inches (8 to 46 cm.) away from the original site of injection.

CLEVELAND DERMATOLOGICAL SOCIETY

JOHN A. GAMMEL, M.D., *President*

Oct. 27, 1938

C. G. LAROCCO, M.D., *Secretary*

J. R. DRIVER, M.D., *Reporter*

Lupus Vulgaris. Presented by DR. H. N. COLE and DR. J. R. DRIVER.

C. M. C., a Negress aged 43, states that between the ages of 3 and 15 she had suppurating cervical glands which were incised on numerous occasions. Following this an eruption developed on the left side of the face and ear, which has slowly progressed. During the succeeding period, of more than twenty-five years, she has received no treatment of any kind, and her general health has been good.

On the left side of the face and the left ear there is a dull red granulomatous lesion consisting of a serpiginous eruption larger than a palm with nodular borders and a scarring center. Under diascopic pressure apple jelly tubercles can be demonstrated. There was no evidence of pulmonary tuberculosis on physical examination.

Serologic tests for syphilis were negative.

Histologic examination of tissue from the edge of a nodule showed the epithelium to be thin, with some parakeratosis. The normal arrangement of the rete pegs was disturbed by an infiltrate which extended from the corium up to the basement membrane. This infiltrate consisted largely of round cells, mononuclears and epithelioid cells. There were poorly formed giant cells and areas of necrosis. Occasional areas showed a few plasma cells and eosinophils.

DISCUSSION

DR. HAL ELSON FREEMAN: I should like to ask how frequently lupus vulgaris is seen in Negroes.

DR. C. L. CUMMER: The impression I have is that it is fairly frequent, while lupus erythematosus is rather infrequent.

DR. JOHN A. GAMMEL: Lupus vulgaris is much more frequent in Europe than in this country, and I think more frequent in white persons than in Negroes. Comparatively few white persons acquire lupus vulgaris in the United States. On close questioning one usually finds a patient to be an immigrant.

DR. J. R. DRIVER: The interesting thing about this particular patient is that she has had tuberculosis of the cervical glands practically since infancy and had numerous incisions in childhood for these nodes. This process has been going on since she was a small child. It is no doubt secondary to tuberculous adenitis. It is remarkable that no treatment has been given in the more than twenty-five years that she has had lupus vulgaris.

Congenital Syphilis with Nerve Deafness after Therapeutic Malaria and Chemotherapy. Presented by DR. H. H. JOHNSON (from the Department of Dermatology and Syphilology, Lakeside Hospital).

E. Z., a woman aged 29, first came under observation in July 1931. She complained of blurring of vision and pain in the right eye for three weeks. Examination at that time revealed characteristic syphilitic interstitial keratitis of the right eye, prominent frontal bosses and rhagades about the mouth. The Wassermann and the Kline reaction were strongly positive. The spinal fluid was normal. The patient had seven malarial paroxysms. There was marked improvement of the keratitis. From August 1931 to March 1934 she received thirty-three injections of neoarsphenamine and forty-five injections of bismuth subsalicylate in alternating

courses. The Wassermann reaction of the blood remained strongly positive throughout therapy. The patient then lapsed from treatment.

She returned on Sept. 27, 1938, complaining of tinnitus in the left ear for six months and deafness for two months.

The left cornea shows a slight opacity, and there is a decided opacity of the right cornea, with no evidence of activity. The pupils react normally to light and in accommodation. The tympanic membranes are slightly retracted. Weber's test was negative. Rinne's test showed air conduction greater than bone conduction bilaterally. With the tuning forks the high tones were absent bilaterally. The spoken voice is heard with difficulty. The upper central incisors are spaced and notched.

The patient has been placed on biweekly injections of 2 cc. of iodobismitol with saligenin.

Erythroderma of the Hands and Wrists in a Syphilitic Patient. Presented by Dr. Frank McDonald (from the Department of Dermatology and Syphilology, Lakeside Hospital).

M. K., a woman aged 58, states that after an appendectomy nineteen and one-half years ago diffuse redness of her hands and wrists developed and has continued ever since. There are no subjective complaints except from a cosmetic point of view. She gave no history of syphilitic infection and had received no treatment for the disease until the past three weeks, during which she has received five intramuscular injections of iodobismitol with saligenin. The Wassermann and the Kline test were strongly positive on two occasions just prior to the institution of this therapy. The spinal fluid was normal.

Symmetrically on both hands and wrists there is an atrophic and slightly scaling reddish violet eruption, not raised above the surface of the skin. In the central portions of the palms there are scaling and considerable atrophy. On the right calf there is a similar lesion, fairly circinate and about 5 cm. in diameter.

General physical examination showed arteriosclerotic changes in the fundi of the eyes and moderately uniform cardiac enlargement with distant heart sounds. There was moderate dyspnea on exertion. The blood pressure was 140 systolic and 100 diastolic. Urinalysis revealed a trace of albumin. Roentgenograms of the chest showed normal pulmonary fields.

A section from the periphery of the lesion on the right wrist revealed an intact layer of stratified squamous epithelium with slight atrophy of the epidermis. The granular layer was prominent, and the rete pegs in the cutis were inconspicuous. In the corium there was an increase in the amount of connective tissue, and a moderate number of fibroblasts were seen. There were focal collections of small round cells, mostly lymphocytes. This infiltrate was practically all in the papillary layer. Some round cells had migrated into the epidermis. Special stains failed to reveal the presence of spirochetes.

DISCUSSION

Dr. H. G. Miskjian: If it is proposed by the presenter that this case is one of syphilitic erythroderma, he must know of other cases that have been described. Erythroderma in late syphilis is known to exist. It comes especially on the thighs and hips. It has nothing to do, however, with this type of lesion. Consequently, I cannot agree with the diagnosis as presented. I have nothing definite to offer except a suggestion based on the erythema and atrophy. Because of the symmetry, atrophy and redness, I am inclined to think this condition may be idiopathic atrophy of the skin. The patch on the right calf speaks in favor of that diagnosis also.

Dr. J. R. Driver: If this is syphilis, it is different from any syphilitic lesion I have ever seen. The improvement apparent from comparing the photograph and the lesion as it is today is definite, and the patient has received antisyphilitic therapy for about three weeks. That would perhaps favor a diagnosis of syphilis.

I have never seen areas of atrophic lichen planus as large as these, but there is something about the appearance of the atrophy—the dry, scaly parchment-like character of the atrophic area—that suggests atrophic lichen planus. The pathologic picture with an infiltrate in the subpapillary layer also suggested lichen planus. A certain amount of heavy metal treatment might have caused the improvement that seems to have occurred. I favor a diagnosis of atrophic lichen planus, for the present at least.

DR. JOHN A. GAMMEL: I saw the patient when she first presented herself at the clinic, and I am not sure that there is such striking improvement.

DR. H. J. PARKHURST, Toledo, Ohio: I am inclined to agree with Dr. Miskjian.

DR. J. EDGAR FISHER: I think the whole process has been caused by some vascular changes due to syphilis.

DR. FRANK MCDONALD: Review of this slide left no doubt that the picture did not fit into what is usually expected in late syphilitic lesions. The points of interest seemed to be the atrophy, the fact that almost all the cellular infiltration was in the subpapillary layer and the fact that the granular layer appeared to be definitely prominent. In addition there were less fibroblasts than would be expected in a syphilitic process and a lack of perivasculitis. I only suggested the possible syphilitic origin of the disease because the patient is syphilitic and the condition has definitely improved on heavy metal therapy. According to the patient, never since the onset have the lesions been as pale as they now are.

DR. E. W. NETHERTON: Were there any neurologic abnormalities?

DR. FRANK MCDONALD: None.

Idiopathic Lymphedema of the Upper Lip. Presented by DR. FRANK MCDONALD (from the Department of Dermatology and Syphilology, Lakeside Hospital).

E. D., a well nourished girl aged 15, states that four years ago she had a furuncle on the upper lip, at the orifice of the left nostril. Since then there have been many exacerbations and remissions of the swelling, but at no time has the lip returned to its original size. The swelling has been present without remission for nearly a year. On the left side it is more prominent. There is little or no demonstrable pitting on pressure. There is a moderate degree of puffiness of the lower eyelids.

Routine examinations of the blood and urine gave negative results. Tuberculin tests were negative, and a roentgenogram of the chest was normal. The basal metabolic rate was —18 per cent.

The following treatment produced no benefit: epinephrine administered on two occasions, daily doses of 2 grains (0.13 Gm.) of thyroid, four doses of 100 roentgens each of roentgen rays and short wave diathermy.

DISCUSSION

DR. EMERSON GILLESPIE, Canton, Ohio: The history would suggest a streptococcic infection as the cause of the trouble. The hardness of the tissues sometimes seen is not present.

A Case for Diagnosis (Dermatitis Herpetiformis or Vesicular Lichen Planus?). Presented by DR. W. F. SCHWARTZ (from the Department of Dermatology and Syphilology, City Hospital).

D. L., a white girl aged 7½ years, has an eruption consisting of poorly marginated patches of lichenified dermatitis with excoriated, vesicular borders, which developed about one year ago. The areas average from 8 to 12 cm. in diameter and are located on the upper and outer portions of the buttocks and the sacrum. The process has persisted, and is associated with marked pruritus. Considerable pigmentation surrounds the lesions.

Histologic examination showed an irregular surface, mild hyperkeratosis and hypergranulosis. The epidermis was of approximately normal thickness in most places, but showed slight acanthosis. Scattered lymphocytes and large mononuclear cells were present about blood vessels and the glands of both the superficial and the deep portion of the corium.

DISCUSSION

DR. H. G. MISKJIAN: The lesions are red and the size of half a dollar or larger and show evidence of a vesicular reaction. The process is a definite dermatitis of infectious origin. The fact that it has been of long duration is not entirely against a diagnosis of a streptococcic infection, for such a process may persist a long time. Against a diagnosis of contact dermatitis is the presence of circinate lesions.

DR. H. J. PARKHURST, Toledo, Ohio: I do not see anything to suggest any form of lichen planus with which I am familiar. I think that the localization of the eruption is against dermatitis herpetiformis. In a patient who is a bed wetter it might be due to contact with decomposition products of the urine. Or the process may be toilet seat dermatitis. I have seen similar lesions in children following repeated contact with toilet seats to which saponated solution of cresol had been applied.

DR. E. W. NETHERTON: I agree with Dr. Parkhurst. The possibility of toilet seat dermatitis is strong.

DR. C. L. CUMMER: In the cases I described of toilet seat dermatitis due to dye or stain (ARCH. DERMAT. & SYPH. **27**:976 [June] 1933) there was an area showing exactly where the patient had sat, which I do not think can be seen in this case. There is almost a saddle mark on the body over the region of the sacrum. I do not see why this cannot be explained as a chronic infectious eczematoid dermatitis.

DR. GEORGE HASKEL CURTIS (by invitation): I questioned the little girl and her mother rather closely about this eruption. It has been rather constant for about a year. There has never been a period in which it improved or got worse. The toilet seat in their home is of white enamel, and it would be extremely rare for white enamel to cause a dermatitis. The patient, on the other hand, played considerably in the playgrounds, and both she and her mother think the condition may have been worse after school started. They say the toilets are painted brown in school. I suggest that an investigation be made and some of the scrapings from the school toilets used in patch tests.

DR. E. J. ARDAY, Lakewood, Ohio (by invitation): I found recently a new cause for toilet seat dermatitis in a baby who is using a toilet seat covered with a rubberized sheet. I performed a patch test with the substance, and the result was positive.

DR. W. F. SCHWARTZ: My associates and I were satisfied in our own minds that this eruption was not a contact dermatitis. The areas of intervening normal skin argue against such a diagnosis. We have been somewhat in doubt, and I presented the case with a questionable diagnosis to stimulate discussion.

Three Cases of Insect Bites. Presented by DR. W. F. SCHWARTZ (from the Department of Dermatology and Syphilology, City Hospital).

A mother and 2 children present erythematopapular lesions on the lower extremities of four weeks' duration. Many of the lesions are equidistant from each other and are arranged in straight lines, circles and various geometric figures. Some show a suggestive central punctum. The family moved into their present house about eight weeks ago.

DISCUSSION

DR. C. L. BASKIN, Akron, Ohio.: I do not think these cases need much discussion. The lesions are bites from cat fleas. Dog fleas seldom bite. If the

patients put on white stockings and sit around in the dark and the physician turns on the lights quickly, he will find the fleas. As a species of fleas they are tiny. The mother states that she has seen little black bugs that fly, but the difference between flying and hopping depends on the patient's mentality. This test is simple and invariably results in clearing up what may otherwise present a problem in diagnosis.

DR. H. A. HAYNES: I have had an experience recently with cat fleas. I had a cat, which I have got rid of, but the fleas got in the carpets and it was finally necessary to have the entire house fumigated in order to get rid of the pests.

DR. J. R. DRIVER: Insect bites often present a problem in diagnosis. In the cases presented today the grouping of the lesions was characteristic. Dr. Baskin's suggested method for the detection of fleas is probably not generally known by dermatologists.

NOTE.—After following the suggestion of Dr. Baskin the presenter reported that the offending insects were easily demonstrated and were identified as cat fleas.

Partially Differentiated Adenocarcinoma of the Stomach with Multiple Less Well Differentiated Metastases to the Skin. Presented by DR. W. F. SCHWARTZ (from the Department of Dermatology and Syphilology, City Hospital).

A. S., a moderately emaciated pale man aged 64, was apparently well until two years ago, at which time he began to complain of vague epigastric distress. This has become gradually worse, and there have been associated anorexia and loss of 20 pounds (9.1 Kg.). He began to notice a few "lumps" under the skin approximately twelve months ago. In the past several weeks these subcutaneous nodules have rapidly increased in number, but the earlier ones have changed little in size. Two and one-half weeks ago an exploratory laparotomy was done and a specimen taken from an 8 cm. mass in the wall of the stomach near the pylorus and another from a mesenteric lymph node. Histologically the tumor was a partially differentiated adenocarcinoma.

Over the upper half of the trunk and on the neck are several hundred shotty subcutaneous nodules, measuring up to 8 mm. in diameter. They are discrete, firm and freely movable, without noticeable involvement of the overlying epidermis. There is no lymphadenopathy.

Roentgen examination of the chest showed the lungs to be free of metastatic lesions. Roentgenograms of the stomach showed a large filling defect at the lower half of the greater curvature.

Histologic examination of a subcutaneous nodule showed the epidermis to be thin, irregular and without other significant changes. Situated deep in the corium and subcutaneous tissue there were irregular, poorly circumscribed collections of abnormal cells. These cells varied greatly in size, shape and character. Generally they were spindle shaped, with deep-staining vesicular compact oval or irregularly shaped nuclei and moderately abundant fibrillary or finely granular cytoplasm. The cells failed to form any histologic pattern, but as seen in azocarmine preparations, formed a fine network of new collagen. Stains for fat showed no lipoid in the tumor cells. About the tumor cells there were a few inflammatory cells, chiefly lymphocytes and large mononuclear leukocytes.

DISCUSSION

DR. W. F. SCHWARTZ: Before the exploratory laparotomy was performed and the character of the primary tumor determined, there was considerable speculation concerning the diagnosis of the widespread cutaneous and subcutaneous nodules. Clinically my associates and I considered metastatic carcinoma, leukaemia cutis and sarcoid. It is unusual for a generalized carcinomatosis such as this to have lesions lasting now for a year or more. Life expectancy in such cases is generally a matter of a few days or a few weeks at most. It is well known that carcinoma

of the gastrointestinal tract results in extensive cutaneous metastases more frequently than carcinoma seen in any other location.

Sarcoid-Like Tuberculosis Cutis Accompanied with Unusual Osteosclerotic Changes. Presented by DR. H. H. JOHNSON (from the Department of Dermatology and Syphilology, Lakeside Hospital).

R. W., a well developed and well nourished Negro aged 30, in April 1925 underwent pericardiotomy for acute fibrinous pericarditis. Endocarditis with aortic insufficiency developed at that time. In 1929 he noticed red nummular lesions on the shaft of the penis, and three weeks later similar lesions appeared at the base of the penis, extending circularly around the shaft in seven weeks. One month later he noticed a number of nodules on the scrotum and adjacent aspect of the left thigh and one on the back of the neck. Since January 1931 the patient has had pain in the spine, sacrum and iliac bones. All the lesions became depigmented and have remained unchanged to date.

At the present time, encircling the shaft of the penis are two irregular, sharply marginated, slightly elevated pale red plaques, with dry, scaling surfaces. These areas are slightly thickened and firm, with some loss of elasticity. They are not adherent to the underlying structures. On the scrotum are about twenty-five nodules of similar color and consistency. On the inner aspect of the right thigh and on the back of the neck are irregular-shaped nodular areas of the same type.

The heart was enlarged, with physical evidence of aortic insufficiency. There were marked limitation of movement of the spine and tenderness on palpation over the dorsal vertebrae and over both sacroiliac joints. Urinalysis and blood counts showed no abnormality. Serologic tests for syphilis were negative. The tuberculin test with a dilution of 1:10,000 was negative, while with a dilution of 1:1,000 it was positive. The serum calcium content was 10.2 mg. per hundred cubic centimeters, the serum phosphorus 5.3 mg. and the serum phosphate 21 units.

Roentgenograms of the bones in 1931 revealed localized sharply defined rounded confluent areas of increased density in the ilia, sacrum and lumbar portion of the spine. Repeated subsequent roentgenograms have shown slow progression and extension of the osteosclerotic process to involve the scapulas and the lower ribs. Roentgenograms of the lungs were repeatedly normal.

A histologic examination of the ilium showed unusually compact spongy bone. The marrow was fibrous; in many fields there were granulomatous lesions with central giant cell formation of peripheral epithelioid cells and dense mononuclear infiltration. A diagnosis of tuberculous osteomyelitis was made.

Histologic examination of a lesion on the penis showed considerable depigmentation of the basal layer of the epithelium, beneath which was granulomatous infiltration in the upper part of the corium. There were numerous typical tubercles with central caseations, epithelioid cells, Langhans giant cells and lymphocytic infiltration.

DISCUSSION

DR. E. W. NETHERTON: Several years ago I presented this patient at a meeting of this society with a diagnosis of sarcoid. There was general agreement on the diagnosis at that time. The histologic picture was that of sarcoid, and there was no typical tuberculosis as described today. Also there was an anergy to tuberculin at that time. The bony changes have increased in intensity. The lesions are definitely atrophied, and there is some evidence of more scarring. I now feel that this case is one of a type described in the recent literature as sarcoid-like tuberculosis of the skin.

DR. JOHN E. RAUSCHKOLB: This case resembles in many particulars a case that was carefully studied at the City Hospital a few years ago. Dr. Fred Weidman saw the patient and stated that he believed the condition the same as that described by him as sarcoid-like tuberculosis of the skin. The histologic structure of a lesion of the skin was the same as that shown today. Pulmonary tuberculosis subsequently developed, and I believe the patient is at present in the tuberculosis division of the City Hospital.

BROOKLYN DERMATOLOGICAL SOCIETY

MORTIMER J. CANTOR, M.D., *President*

Nov. 11 and Dec. 19, 1938 and Jan. 16, 1939

SEYMOUR H. SILVERS, M.D., *Secretary*

Lupus Erythematosus Disseminatus in a Young Boy. Presented by DR. M. DICKMAN.

R. A., a boy aged 11 years, a Puerto Rican, is presented from the Jewish Hospital because of an eruption of about eleven months' duration. It consists of numerous well circumscribed pea-sized and smaller depigmented flat lesions with surrounding hyperpigmentation and is located on the bridge of the nose and cheeks in a butterfly arrangement. Some of the lesions show atrophy, and in others there is a suggestion of atrophy. There are similar lesions on the ears and arms, most of which are atrophic. Intermingled with those are a few small erythematous slightly raised lesions with small white adherent scales.

There is no history of tuberculosis in the family. A roentgenogram of the chest a few months ago revealed that the pulmonary fields were equal in size and within normal limits, and a calcified Ghon's tubercle was demonstrated in the base of the right lung. A Mantoux test with 0.1 mg. of tuberculin gave a strongly positive result. The Kline test was negative. A physical examination showed no abnormality. Histologic examination of a specimen removed from the arm showed evidence of an acute inflammatory process, with no basophilic degeneration or change in the elastic tissue.

DISCUSSION

DR. DAVID M. DAVIDSON: Because of the small erythematous patches with adherent scales and definite atrophic spots on the face, I think this case is one of lupus erythematosus of the disseminated variety. However, the histologic picture did not corroborate the diagnosis.

DR. JACOB SKEER: When the patient first came to the clinic he showed marked clinical symptoms of what in my opinion was subacute lupus erythematosus. The improvement has been great, but there is still clinical evidence of atrophy.

DR. L. FRUCHTBAUM: I should like to ask whether there were constitutional complaints before or during the time of the eruption. Usually with acute disease there is a febrile reaction, particularly in children. The condition is usually serious, but this boy does not look sick. However, the present appearance of the lesions is that of lupus erythematosus. There is definite atrophy, and the distribution is characteristic. I think the condition is a "fixed" type of lupus erythematosus.

DR. DAVID L. SATENSTEIN: On clinical grounds I cannot accept the diagnosis of lupus erythematosus at the present time. The members will have to accept the presenter's statement that the condition was lupus erythematosus. Exactly what type it was I do not know. The acute type never starts with macular lesions like these. There are usually erythematous areas, and the patients are very sick as a rule. This patient presents depigmented and hyperpigmented areas on the cheeks and one atrophic area on the bridge of the nose. There is some vague scaling on the ears, which I was told was an active area. The microscopic picture did not suggest lupus erythematosus but a simple dermatitis.

DR. L. J. FRANK: I am inclined to agree with Dr. Satenstein. If this boy had acute lupus erythematosus I think he would be very sick. He does not give a history of being sick at any time. Therefore this condition will have to be placed with the discoid type of lupus erythematosus.

It seems that there is a tendency to regard any red or scaly cutaneous lesions on the face as lupus erythematosus. I am under the impression that lupus erythematosus is a disease of the young adult and is rare in children.

DR. MORTIMER J. CANTOR: Lupus erythematosus was excluded by microscopic examination. In view of the negative microscopic picture and the present clinical picture, I suggest a toxic dermatosis, rather than definite lupus erythematosus.

DR. M. DICKMAN: This case was not presented as one of lupus erythematosus disseminatus acutus. The condition belongs to the subacute type which may become discoid. Concerning the age of this patient, it is a fact that lupus erythematosus is not common in chidren, but it does occur, nevertheless. C. L. Cummer in his article on lupus erythematosus in children (ARCH. DERMAT. & SYPH. **24**:999 [Dec.] 1931) stated that only about 4 per cent of patients are children up to 15 years of age. I believe this case to be one of superficial erythematous type of lupus erythematosus, apparently clearing with residual mild atrophy.

Alopecia Atrophicans (Pseudopelade of Brocq). Presented by DR. SEYMOUR H. SILVERS.

J. B., a single woman aged 25, doing housework, is presented from the Wyckoff Heights Hospital. She complains of bald spots on the scalp of six years' duration. She has always been well, and 6 brothers and sisters and her father and mother are living and well. No one in her immediate family is similarly affected.

Since the onset of this disease, six years ago, the areas have gradually become larger. There were never any "pimples" or "bumps."

The Wassermann reaction was reported negative, and examination of the urine showed nothing abnormal.

Scattered over the scalp are numerous well defined oval and round shiny atrophic areas varying in size from that of a penny to that of a silver quarter and devoid of hair and hair follicles. Isolated hairs can be seen at the border of the lesions. The surrounding skin and hair follicles show no evidence of inflammation. The scalp shows no other evidence of disease.

DISCUSSION

DR. DAVID L. SATENSTEIN: This condition is progressive in the sense that new foci appear. Once the atrophic areas appear they do not get larger, but they may coalesce. I think it belongs to the primary atrophies of the skin, similar to atrophy on the body. The histologic structure is identical. I have had an opportunity to study a number of cases. I found that if the same process was present elsewhere there was no difficulty in diagnosis. Regarding preceding inflammatory reactions, I have observed a number of cases in which no inflammation of the skin was present. I think this case belongs in that group.

DR. SEYMOUR H. SILVERS: In presenting this case, which offers no difficulty in diagnosis, I had certain problems in mind. Atrophic areas of the scalp are seen, yet they are seldom grouped with the primary atrophies of the skin. This atrophy is not preceded by any sign of an inflammatory process. The cause has not been ascertained, but many theories have been advanced. Among these has been the theory of an endocrine imbalance, and cases have been presented in which thyroid therapy helped. Some authors have tried to link this disease with syphilis, leprosy and tuberculosis. Years ago Sabouraud claimed that micro-organisms were the cause. Tibor Benedek has again advanced the hypothesis that parasites are responsible. None of these suggestions, however, has been corroborated. An interesting phenomenon of this disease is that the patches of alopecia are self limited; to my knowledge there has never been complete alopecia due to this disease. I was glad to hear that Dr. Satenstein classifies this condition with the primary atrophies of the skin.

Pityriasis Rubra Pilaris. Presented by DR. C. THOMAS CHIARAMONTE.

This patient, a youth aged 18, born in Italy and apparently healthy, has been handling wax in a candle factory, where he has been employed for the past three years. The past and the family history are noncontributory.

Six months ago he first noticed roughness of the fingers, which gradually extended up the forearms to his elbows. There have never been any subjective symptoms.

On the dorsa of the fingers at the first and second phalanges is a grater-like roughness, produced by the presence of dry scaly conelike papules, through the blackish center of each of which a hair emerges. Similar areas are evident on the dorsa of both hands, around the wrists and extending up both forearms on the volar surface to the elbows. Here the affected areas show a definitely rounded contour, merging in places. The eruption is symmetric.

There are numerous discrete flesh-colored small follicular papules on the sides of the neck, symmetrically situated and extending upward from both supraclavicular fossae.

Hyperkeratosis of both palms is pronounced, but the soles are unaffected.

The basal metabolic rate was —14 per cent. A roentgenogram of the chest revealed accentuation of the upper bronchial radicles extending into the upper lobes of both lungs, indicative of bronchial productive inflammation.

DISCUSSION

DR. L. J. FRANK: I think that the hair follicles are simply plugged with grease. The condition happens to be more noticeable on the backs of the fingers, but the follicles in this location are normally more numerous and larger. I do not agree with the diagnosis of pityriasis rubra pilaris, as there are no seborrheic lesions of the face with scaling, but there is some scaling around the hair follicles on the backs of the fingers and on other parts of the body. In early pityriasis rubra pilaris there should be some scaling on the palms. This patient does not show any erythema. Consequently, I feel that he has keratosis pilaris, and the etiologic factor is some wax or grease which he handles.

DR. L. FRUCHTBAUM: Personally, I do not see the characteristics of pityriasis rubra pilaris. In the first place, there are no lesions on the palms, except those resulting from wax, which are occupational. There are none of the changes in the nails usually found in pityriasis rubra pilaris. There are no patches on the body except a few lesions on the forearms. There is no change on the face and head. The color of the lesions is not that of pityriasis rubra pilaris. The only evidence is the presence of some black dots in the original lesions and the distribution on the proximal and middle fingers. However, on these grounds alone I should not make a diagnosis of pityriasis rubra pilaris.

DR. M. EDWARD GOEBEL: While it is true that all the features of pityriasis rubra pilaris, such as seborrhea, are not present, the coalescing of the papules forming grayish red scaly patches makes me think of early pityriasis rubra pilaris. This is suggested by the hard dry papules at the mouths of the hair follicles. The condition has to have an onset sometime. It is possible that the patient is in the first stage of pityriasis rubra pilaris.

DR. DAVID M. DAVIDSON: I am inclined to consider this case as one of early pityriasis rubra pilaris. I do not agree that the disease is keratosis pilaris.

DR. JACOB SKEER: I am inclined to agree with the presenter that it is pityriasis rubra pilaris.

DR. SEYMOUR H. SILVERS: I think that in this case there are some interesting problems, both of a theoretic and of a practical nature. Pityriasis rubra pilaris is rare, and one observes only a few cases annually, even at the large centers. It is difficult to make a positive diagnosis of this disease if the lesions are limited in extent. This patient shows lesions only on the dorsa of the hands and forearms. In these areas there are pinhead-sized slightly red keratotic follicular papules. The patient gives a history of having worked with wax and oil, substances which are known to produce follicular eruptions in workers. I should therefore suggest that he stop work for two or three weeks. If these lesions are due to his occupation, they should clear or be greatly improved. If they are those of pityriasis rubra pilaris, there will be no change.

DR. C. THOMAS CHIARAMONTE: This boy was sent to me by his employer for treatment as a subject for compensation. I fail to see anything compensable in this dermatosis, and I am finding it difficult to convince the employer that the eruption was not caused by wax. The patient did the same kind of work for two and a half years before the onset of the eruption. He always wears long sleeves at work. The lesions have been asymptomatic from the beginning. There has never been any vesiculation or moisture. The patches on the forearms have a well defined circinate outline. I believe that these facts tend to eliminate the possibility that the disease is occupational.

R. Prosser White (The Dermatergoses or Occupational Affections of the Skin, New York, Paul B. Hoeber, Inc., 1929) mentioned the fact that there has never been recorded a single instance of occupational dermatosis arising from the handling of wax in the candle industry.

I believe that the noteworthy and atypical feature of this case as it is presented this evening is the complete absence on the face and scalp of any seborrhoea sicca-like lesions.

Multiple Idiopathic Hemorrhagic Sarcoma of Kaposi. Presented by DR. DAVID M. DAVIDSON.

C. N., a Jew aged 60, born in Russia, is presented from the Trinity Hospital. The patient has had the dermatosis on the upper and lower extremities for about one year. A detailed history was not obtainable.

On the dorsa of both hands and the proximal phalanx of the right ring finger and of the left index finger are small marginated slightly elevated erythemato-violaceous infiltrated plaques. Most of the color disappears on pressure. On the entire dorsum of each foot and toes, in the region of the left external malleolus and on the anterior aspect of the left ankle are similar infiltrated plaques. The right ankle is enlarged in circumference, owing to a pitting edema, and is surrounded by an erythematoviolaceous patch. On the anterolateral aspect of the lower part of the right leg there is an irregular firm elevated and sharply marginated lavender plaque. The color does not disappear on pressure. A similar firm lavender plaque is found on the posterointernal aspect of the same leg, with a pea-sized depressed scar of the same color. A flat irregular purpura-like patch is present below the left elbow. Erythematoscaly patches of an eczematous eruption are noted among the lesions just described, chiefly on the hands, ankles and lower part of the right leg.

Microscopic examination of tissue from the dorsum of the left hand and the right leg showed Kaposi's sarcoma in the inflammatory stage.

DISCUSSION

DR. DAID L. SATENSTEIN: The only thing I should like to call attention to is a feature which developed since the presentation at the last meeting. Some of the members noticed the erythema around the man's elbow; all that has developed during the past week. In other words, here is an early stage of Kaposi's sarcoma which is probably progressive and taking on the inflammatory type. It is possible that if he survives long enough the other changes will develop.

DR. L. FRUCHTBAUM: I should like to ask Dr. Satenstein whether the taking of tissue could account for the flare-up of the process. Some years ago I had a case of classic disseminated Kaposi's sarcoma on the body, and after I performed a biopsy many new lesions appeared.

DR. DAVID L. SATENSTEIN: The piece of tissue was taken from the right leg, and as the flare-up was on the left elbow, I do not think there could be any connection. If the disease were in the tumor stage it would be possible that the tumor was irritated, causing the pathologic process to become more active. I have 40 to 50 specimens of Kaposi's sarcoma, and I do not remember a single instance in which removal of a biopsy specimen had anything to do with the spreading of the disease. One patient came to me with a tumor mass as big as

my fist on his sole. I removed it by electrodesiccation and the base healed nicely. There has been no dissemination. Taking tissue from a malignant process does it no harm, provided the tissue is not traumatized. With the modern method of removing biopsy specimens this does not happen.

DR. DAVID M. DAVIDSON: This case was previously presented (not published) at the last meeting as one of Kaposi's pigmented hemorrhagic sarcoma. The diagnosis was not accepted by the majority of the members. Microscopic studies since then corroborated the diagnosis.

The fear that one experiences when there is a question of taking a biopsy specimen from malignant or semimalignant growths is due to the knowledge that such an operative procedure may precipitate activity or metastases. Kaposi's sarcoma, according to the present concept, does not metastasize; the lesions found in the internal organs are primary and may appear earlier than, simultaneously with or later than those of the skin.

Folliculitis Ulerythematosa Reticulata. Presented by DR. DAVID M. DAVIDSON.

W. M., a woman aged 31, presented herself at the clinic of the Jewish Hospital with an eruption on her face of three years' duration. It began, according to her statement, as erythematous papules in the preauricular regions.

At present the eruption is symmetrically distributed and consists of a number of erythematous patches covering the forehead, preauricular regions, nasolabial folds and anterior and inferior aspects of the chin. Each patch produces a honeycomb appearance, being composed of closely crowded areas of atrophy the size of a small pinhead and tiny papules. Under diascopic pressure the papules are seen as yellowish brown spots. Telangiectasia is noted on the erythematous patches, and a moderate number of papulopustules are scattered over these areas.

DISCUSSION

DR. L. FRUCHTBAUM: How should this condition be classified? Shall it be put in a group with lupus erythematosus, with the tuberculous diseases or with the infectious group?

DR. C. THOMAS CHIARAMONTE: The lesions usually described for this condition do not conform, according to my opinion, to those in this case. The lesions and the depressions are not typical for this condition.

DR. DAVID L. SATENSTEIN: This disease is just reticulated atrophy with erythema; I cannot see the follicular part as described by the presenter.

DR. JACOB SKEER: The picture as presented may well fit in with the condition known as folliculitis ulerythematosa reticulata, except that the ridges are not as well defined as those which other writers have described. A little more erythema and telangiectasia would lead me to think of Lewandowsky's tuberculid. Another diagnosis that could be considered is lupus miliaris disseminatus faciei, but I cannot see any necrotic nodules.

DR. A. GRACE, New York: I do not believe from the appearance of the patient tonight that a definite diagnosis can be made. If a piece of tissue were excised and examined by a competent pathologist, a diagnosis could be made more readily.

DR. DAVID M. DAVIDSON: This patient was seen only once, and I have no histologic report at present. Clinically, the eruption might suggest one of the following conditions: Civatte's poikiloderma, Lewandowsky's tuberculid and folliculitis ulerythematosa reticulata. Civatte's poikiloderma, however, can be excluded on account of the papulofollicular atrophy and almost complete absence of pigmentation. Lewandowsky's tuberculid has most of the features here present but not, as far as I know, this peculiar atrophy. On the other hand, all the features shown by these erythematous patches together with their symmetric distribution fit in with the condition described as folliculitis ulerythematosa reticulata as nearly as any.

Beginning Primary Syphilitic Atrophy of the Right Eye. Presented by Dr. Samuel Hecht.

C. N., a man aged 32, has a wife and a child aged 5 years. They are well as far as he knows.

The patient had a penile sore in 1929. No dark field examination was done, but the Wassermann reaction at that time was negative. He was told that the condition was trifling, and therefore he never took treatment. The rest of his history is irrelevant.

In September 1938 he had difficulty with the vision of his right eye. He came to the New York Polyclinic Medical School and Hospital where an examination showed beginning optic atrophy. The diagnosis was corroborated by the ophthalmologic department. The right pupil was dilated; the right fundus showed general pallor, with a narrow area of white in the temporal region.

The Wassermann reaction of the blood and of the spinal fluid were 4 plus. There were no other neurologic symptoms or signs.

On Dec. 4, 1938, fever treatments with typhoid vaccine intravenously were begun. He was given eight of these, the temperature being elevated to from 104 to 106 F. at each. As a result of treatment, there was at least 75 per cent improvement.

DISCUSSION

Dr. David L. Satenstein: I should like to know just what was meant when the presenter stated that in the atrophy there has been 75 per cent improvement. Atrophy is atrophy, and treatment does not change it.

Dr. L. J. Frank: Primary syphilitic atrophy is not uncommon. When optic atrophy develops the patient should be referred to an ophthalmologist for a complete examination and advice as to further treatment. As far as treatment is concerned, I should not use the old arsphenamine. I think that this drug is contraindicated in the treatment of primary atrophy of the eye. I should prefer a milder preparation, such as neoarsphenamine or silver arsphenamine, and use it in small doses. If possible, I should dispense entirely with arsenicals and lean more to the use of heavy metals such as bismuth or mercury preparations.

Dr. Abraham Walzer: What the presenter probably means to imply is that this man had bad vision with signs of optic atrophy which improved under treatment. Some of the loss of vision in all probability was not due to the destruction of the nerve but was caused by pressure symptoms from the inflammatory process in or around the nerve. Treatment cleared the inflammation but not the atrophy. Optic atrophy cannot be influenced by treatment.

Dr. Jacob Skeer: On examining the eyes with the ophthalmoscope, I gained the impression of right temporal pallor. On the left side of the right disk I noticed unusually tortuous vessels. I am in no position to say whether there has been any improvement. It is thought that one can get fairly good results with antisyphilitic therapy in secondary optic atrophy.

Dr. M. L. Weitz, Jamaica, N. Y.: According to the history the patient had a penile lesion in 1929. I want to go back to that time, and what I am trying to do is to criticize the way the patient was handled at that time. The Wassermann reaction at the time the penile lesion was present was apparently reported as negative. I feel that if syphilis had been determined to be present at that time, optic atrophy might never have developed, because there is no doubt that the penile lesion was a chancre. If the patient had repeated Wassermann tests every two weeks, in spite of the fact that no dark field examination was done—or if it was done, it was negative—the presence of syphilis would have been determined, and if the patient had received the necessary treatment this ocular condition would never have developed.

Dr. Samuel Hecht: When this man came to me, his right eye showed decreased vision. Incidentally, I did not rely on myself but sent him to the

ophthalmologic department, which agreed with the diagnosis. I presented him mainly for treatment. I agree that the results in the vast majority of cases of optic atrophy are terrible; most patients go on to complete blindness. On the other hand, if the diagnosis of primary optic atrophy is made early enough one can obtain good results. I feel that fever treatment should be used first, followed by arsenical medication. There was a report before the American Medical Association this year of the use of tryparsamide in the treatment of atrophy of the optic nerve (Leinfelder, P. J.: Pathologic Changes in Amblyopia Following Tryparsamide Therapy, *J. A. M. A.* **111**:1276 [Oct. 1] 1938). I should not have enough nerve to use that drug, as I have seen some pretty bad results from it. I tried other means, but most of them proved failures in most cases. I think that if beginning optic atrophy were treated intensively, good results would be possible in some of the cases. When the results are good they are very good and last a long time.

Dermatitis Lichenoides Chronica Atrophicans (Csillag). Presented by Dr. David M. Davidson.

F. E., a Negro aged 67, is presented from the Trinity Hospital. He observed the first lesion of his eruption nineteen or twenty years ago on the right elbow; the latest lesion, which is on the abdomen, appeared four or five years ago.

At present there is a horizontal patch about 8 inches (20 cm.) long on the lower part of the abdomen; it is erythematous and depigmented, with a circinate and hyperpigmented border. The affected area is somewhat rough and wrinkled; a few dark red spots the size of a pinhead can be seen in the interior of the plaque. This large lesion is formed by the confluence of a few small patches, as shown by the configuration and the hyperpigmented spots scattered over the large patch.

Two small similar patches of oval shape are located below the large lesion. On the left wrist and the right elbow are similar lesions of irregular shape.

A few large dry scales, mostly detached from the skin, were observed on the patches when the patient was first seen, about three weeks ago.

The Wassermann and the Kline test gave negative results.

Microscopic examination showed dermatitis lichenoides chronica atrophicans.

DISCUSSION

Dr. David M. Davidson: Dermatitis lichenoides chronica atrophicans is a rare disease and is worth presenting, especially in a Negro. Clinically, it is often confused with lichen sclerosus et atrophicans (Hallopeau) and scleroderma; therefore microscopic examination is essential. The diagnosis in this case was corroborated by microscopic study.

Dr. C. Thomas Chiaramonte: I was unable to discern any papules within or in the vicinity of the lesion on the lower part of the abdomen, the surface of which was not smooth or atrophic. In view of the duration of the lesion and the presence of cross striations in it, I should prefer a diagnosis of neurodermatitis.

Dr. L. J. Frank: It seemed to me as though there were sharply outlined infiltrated and shining patches. I could not make out any individual papules. The border of the lesion is pigmented, but I think that in most cases of neurodermatitis one would expect to find pigmentation after a while. The excoriations are not uncommon in neurodermatitis. I have not seen many Negroes with dermatitis lichenoides chronica atrophicans. A microscopic examination should help in making the diagnosis. Judging from the clinical aspect, I think this case is one of neurodermatitis.

Dr. Abraham Walzer: I do not think there is any question as to atrophy, and therefore I cannot see how a diagnosis can be made at this stage, when there are no primary lesions. Concerning the histologic report: I was always under the impression that the histologic structure of Csillag's disease was not definite,

and the histologic diagnosis was made only by exclusion of lichen and other conditions. The diagnosis of Csillag's disease is made on a clinical and not a histologic basis.

DR. SEYMOUR H. SILVERS: When I examined this patient I failed to see atrophy. Other examiners, however, were sure that they saw atrophy of the skin. This brings up the question of what are clinically the criteria of cutaneous atrophy. That they are vague is evident from the fact that different examiners at the same time read the skin differently. Dr. Davidson told me that the epidermis looked normal under the microscope.

DR. L. M. WATERHOUSE, New York: I did not see this patient, but Dr. Walzer thought the condition was Csillag's disease. I never have found out precisely what Csillag's disease is. The differentiation between Csillag's disease, lichen albus of von Zumbusch, the guttate type of morphea, lichen planus sclerosus et atrophicans of Hallopeau and lichen morphoicus of Crocker is difficult. I have heard a great many discussions at conferences concerning these diseases, but no one has ever seemed to have any clear idea of the difference between them. I should like to know how to differentiate them.

DR. A. GRACE, New York: It is my impression that this patient has neurodermatitis. I agree with Dr. Chiaramonte that there is no atrophy.

DR. MORTIMER J. CANTOR: I think that the diagnosis should rest on the proper history of the initial lesion, and that is not available. The history does not say what it was like. In view of the fact that the end process is present, it is really impossible to tell what might have been, i. e., lichen planus, morphea, a patch of scleroderma or papules characteristic of Csillag's disease. These entities may end in the same way. Of course the histologic picture right now is important, and I should like to hear from some one who examined the slides.

Acrodermatitis Chronica Atrophicans. Trophic Ulcers. Eczema. Unilateral Enlargement of the Thyroid Gland. Presented by DR. SEYMOUR H. SILVERS.

J. K., a white housewife aged 54, born in Austria, is presented from the Wyckoff Heights Hospital. She complains of ulcerations on the lower part of the left leg of one year's duration. She has always enjoyed good health, has not lost weight, is not excessively nervous, sleeps fairly well and does not suffer from diarrhea. She has given birth to 7 children, 2 of whom died during childhood. The others are living and well. Since early childhood, she has had an enlarged mass in the front portion of the neck. One year ago she received six injections for varicose veins. At the site of one injection a red scaly patch developed.

Examination shows her to be thin, with an orange-sized semisolid mass in the front portion of the neck, right off the midline. The skin of the upper extremities is violaceous and has a soft doughy feeling. The same color and feeling of the skin are present on the upper half of the thighs. The skin of the dorsa of the hands approaching the fingers is thinned. The skin of the feet, legs, knees and lower areas of the thighs is thinned, showing the subcutaneous vessels. The color is bluish to violaceous, and the consistency of the skin is papery. There is a dollar-sized irregular scaly and somewhat crusted red patch on the upper inner side of the left leg. On the lower third of the same leg, in the region of the ankle, there are two pea-sized deep-seated ulcers and the skin is drawn tight and is shiny.

The Wassermann test was reported to be negative. Examination of the urine showed no abnormality.

DISCUSSION

DR. DAVID L. SATENSTEIN: I should like to say something only as a warning: When a patient has acrodermatitis chronica with dilated veins resulting, the veins should be let alone, because the physician will be subject to a lawsuit. The tissue will not stand the damage done, and a great deal of breaking down might ensue.

DR. SEYMOUR H. SILVERS: I believe that this patient has fairly typical acrodermatitis chronica atrophicans.

I am glad that Dr. Satenstein cautioned against thrombosing the veins in treating this disease. When I first saw this patient and obtained the history that she had received injections for varicose veins, I was sure that the physician had missed the diagnosis, and because of this, I took the privilege of showing what is usually considered a typical condition. The areas which now show ulceration and patches of eczema are those where injections were given.

Lichen Spinulosus. Presented by DR. MORTIMER J. CANTOR.

M. J., a boy aged 12 years, American born of Italian parentage, was first seen on Dec. 20, 1938, at the dermatologic clinic of the Beth Moses Hospital. There was nothing of any importance in his past history. The eruption was of about two months' duration.

At present he has a sparsely scattered eruption on the back, chest, upper extremities and nape of the neck, consisting of dime-sized to quarter-sized patches appearing in crops. They are mildly erythematous, enlarge progressively and fade gradually. There are discrete individual papules in addition to those composing the patches. The patches are composed of minute papules situated about the follicles, and many have a projecting horny spine, some barely perceptible to the touch and others protruding about 1/16 inch (0.16 cm.) above the surface of the skin. The shorter type of spine is visible as dark brown or black débris in the follicular opening.

Physical examination revealed a well developed robust boy with no essential abnormality.

Urinalysis gave normal results.

Histologic examination of a section from the supraclavicular patch corroborated the clinical diagnosis.

DISCUSSION

There was no discussion. The diagnosis was unanimously agreed on.

Acrodermatitis Chronica Atrophicans with Pseudosclerodermal Changes. Presented by DR. ABRAHAM WALZER.

F. B., a woman aged 72, has been an inmate of the Jewish Sanitarium and Hospital for Chronic Diseases for four years, because of right hemiplegia and chronic heart disease. The condition of her skin dates back about forty years.

The dermatitis is symmetrically distributed on the backs of the hands, forearms, lower extremities, buttocks and lumbar region. The eruption begins with bright red to dusky red patches of various shapes and sizes. With further progress of the disease, some of these patches become slightly elevated and infiltrated. Ultimately the skin on these areas becomes loose and wrinkled, is easily raised from the underlying tissues and assumes the appearance of wrinkled cigaret paper. The veins in these locations shine through the wrinkled skin, indicating that the skin has been thinned.

On the ulnar side of each forearm is a strip of erythema which shows atrophic areas of skin and extends from the wrist to the elbow. Below the right elbow posteriorly is a dime-sized hard nodule, embedded in and below the skin.

Almost completely surrounding the middle third of each leg, like a cuff, and apparently within the atrophic area, is dirty white induration. It is smooth on the surface, but irregular and not sharply demarcated. It is bordered above and below by atrophic skin. In this area the sensation given to the palpating finger is that the induration is composed of bands of deep irregular infiltration.

On the lower anterior part of each thigh, just above the knee, is a boggy tumor the size of a large fist, surmounted by atrophic skin and easily movable in all directions.

Roentgen examination of the skull, the long and small bones of the extremities, the hip bones and the chest revealed nothing abnormal except mild arthritic changes in some of the smaller joints. In addition, roentgen examination of the lower third of the thighs and legs showed a diffuse network-like spread of calcareous deposits in and under the skin but not in the muscles.

Chemical examinations of the blood, blood counts, examinations of the urine, and determinations of the basal metabolic rate gave results within normal limits. Wassermann tests were negative.

DISCUSSION

DR. JACOB SKEER: This patient has fairly widespread acrodermatitis chronica atrophicans. What interests me most is the pseudoscleroderma condition of the legs. Many cases of calcinosis in association with scleroderma have been reported, but none that I can remember in connection with acrodermatitis chronica atrophicans. It is possible that this case is one of acrodermatitis chronica atrophicans with calcinosis, which is rare.

DR. DAVID M. DAIDSON: Every one here will agree that this patient presents acrodermatitis chronica atrophicans. The interesting point, however, is the calcium deposition in the skin of the legs and thighs, shown by the roentgenograms. The combination of calcium deposits in the tissues and acrodermatitis chronica atrophicans must be rare. The presence of calcium deposits in scleroderma is also rare but is observed now and then. The explanation of its pathogenesis is based on the decreased metabolic activity of the tissues owing to fibrosis and hence to low carbon dioxide tension and the ability of calcium to precipitate in such tissues, which are more alkaline than normal. The normal calcium content of the blood is preserved by the replacement by the action of the parathyroids of the blood's lost calcium with calcium of the bones. I wonder whether this hypothesis can explain also the calcium deposits in cases of acrodermatitis chronica atrophicans.

DR. ABRAHAM WALZER: In about 40 per cent of cases of acrodermatitis chronica atrophicans such areas of pseudoscleroderma are present. Histologic sections of pseudoscleroderma in acrodermatitis chronic atrophicans show a picture not of scleroderma but of acrodermatitis atrophicans. There is atrophy of the elastic tissue plus plasma cell infiltration.

DR. MORTIMER J. CANTOR: I might suggest that perhaps the reason other physicians have not found calcium in so-called pseudoscleroderma or pseudoscleroderma-like areas in acrodermatitis chronica atrophicans is because they seldom take roentgenograms, as Dr. Walzer has done.

DR. ABRAHAM WALZER: It is necessary to distinguish between calcinosis and the presence of calcium deposits. Where there is a suppression of activity, as in the tissue in scleroderma or in dead tissue, there is a natural tendency for calcium to be deposited. This is not due to true calcinosis but just to a local process. There are no changes in the blood calcium in this case, a not infrequent finding also in true calcinosis. This condition is not true calcinosis but a localized process in which calcium has been deposited as a result of some local change.

Parapsoriasis Varioliformis et Lichenoides. Presented by DR. SEYMOUR H. SILVERS.

A. U., a school boy aged 15, is presented from the Wyckoff Heights Hospital, complaining of a generalized eruption, only slightly itchy, of over seven months' duration. He was always well. His twin sister is living and well. There is no history of acute illness or an operation. About seventeen months ago the eruption first appeared all over the body. A doctor suggested the diagnosis of chickenpox. The eruption did not run the usual course for this disease. New lesions appeared constantly, while the older ones formed dry patches.

When presented eight months ago, with a diagnosis of parapsoriasis varioliformis, examination showed him to be oversized and obese, with fair skin and

signs of Fröhlich's syndrome. Scattered over the body were numerous discrete and patchy lesions. These consisted of isolated pea-sized papules and vesicles, appearing singly or in patches, on a slightly inflammatory base. The older lesions were brownish red, scaly and somewhat infiltrated. The patches varied in size from that of a dime to that of a half dollar. New lesions were constantly appearing, while the older ones involuted, leaving pigmentation and faint scars.

Since that time he has received only local therapy with 2 per cent ammoniated mercury ointment and bland lotions. Examination now shows, scattered over the trunk and extremities, patches of papules varying in size from that of a lentil to that of a pea and somewhat infiltrated and thickened. Some are scaly. There are also healed pigmented areas and fine pinhead-sized scars.

The Wassermann reaction was negative. The urine was normal. A microscopic section of an early lesion showed mild parakeratosis, evidence of some degeneration of the cells of the rete pegs, beginning disorganization of the basal layer, vascular dilatation and round cell infiltration of the papillary and subepidermal regions.

DISCUSSION

DR. JACOB SKEER: When the patient was first presented I think I agreed with the diagnosis. At this time there is no evidence of parapsoriasis varioliformis. I also do not note any lichenoid lesions or large erythematous plaques. The lesions tonight, as I see them, are small infiltrated psoriasiform lesions, discrete, with little coalescence and bleeding points on removal of scales. I do not notice healed scars, such as one sees in papulonecrotic tuberculid; some lesions on the forearm show pigmentation.

DR. DAVID M. DAVIDSON: I agree with the diagnosis of parapsoriasis varioliformis, as the small erythematous scaly patches, the lichenoid lesions and the variola-like scars presented by this patient are characteristic of this disease.

DR. SEYMOUR H. SILVERS: Today this patient shows few of the varioliform lesions, but the members may recall that when he was presented eight months ago, he showed few of the lichenoid lesions and a great number of varioliform lesions. The first time I saw the patient was nine months after the eruption first appeared. At present this boy presents more of the characteristics of lichenoid than of varioliformis parapsoriasis.

Multiple Pigmentary and Hairy Nevi. Presented by DR. M. EDWARD GOEBEL.

F. R., a girl aged 5 years, since birth has presented multiple pigmentary and hairy nevi over the back and face and a rather extensive one involving the left hand, forearm and lower third of the arm. It is believed that these nevi are becoming lighter in color.

DISCUSSION

DR. M. EDWARD GOEBEL: This patient is presented not for diagnosis but for suggestions as to therapy. I am open to suggestions.

DR. M. L. WEITZ, Jamaica, N. Y.: I saw this child about one and one-half years ago at the Queens General Hospital, Jamaica, N. Y. Men on the staff of the tumor clinic were called in and we all finally came to the conclusion that nothing should be attempted and that the lesions should be left alone.

DR. MORTIMER J. CANTOR: All the lesions, no doubt, should be left alone.

DR. SEYMOUR H. SILVERS: I have nothing to offer therapeutically in a positive sense. In a negative sense, however, it would be good therapeutics to impress on the patient the fact that at the present state of medical knowledge more harm than good may be done by treatment. Two days ago I was consulted by a patient with a flat blue vascular nevus about the size of a nickel on the side of the nose. There was also a lentil-sized depressed opening, resulting from treatment with radium needles. Two years of treatment and observation left the patient with a worse cosmetic result.

MINNESOTA DERMATOLOGICAL SOCIETY

LOUIS A. BRUNSTING, M.D., *President*

Minneapolis, Dec. 2, 1938

F. W. LYNCH, M.D., *Secretary*

Syphilis. Possible Infectious Arthritis. Presented by DR. S. E. SWEITZER, Minneapolis.

M. G., a white girl aged 14, acquired primary syphilis about two months ago. Soon after her exposure a purulent vaginal discharge developed and did not clear up. She was admitted to the Minneapolis General Hospital one month after exposure, at which time both serologic and dark field examinations showed syphilis; however, repeated cervical and urethral smears showed no sign of gonorrhea. Immediately after the diagnosis of syphilis was made, the patient was given neoarsphenamine intravenously every other day, 0.3 Gm. for three doses and 0.45 Gm. for one dose. Thirty-six hours after her last injection she complained of severe pain, tenderness and swelling in the proximal interphalangeal joints of the right thumb and fifth finger and the left fifth finger. Because of the impossibility of making a positive diagnosis of gonorrheal arthritis, symptomatic therapy with salicylates and local heat was instituted. The condition of the joints improved but did not clear up completely. There were scattered petechiae over the left wrist and forearm at the onset of the arthritis, and because of the possibility that these were a reaction to neoarsphenamine, intramuscular bismuth therapy was begun.

There are marked tenderness and some swelling over the proximal interphalangeal joints of the left thumb and fifth finger and some tenderness of the same joint on the right fifth finger. Motion is limited in these joints, and the involved fingers are held in the semiflexed position.

Roentgenograms showed destructive arthritis of the proximal interphalangeal joints of the right thumb and fifth finger. The complement fixation test for gonorrhea was negative.

DISCUSSION

DR. F. T. BECKER, Duluth (by invitation): The patient gave a history of repeated sore throat accompanied with migratory articular symptoms. This would lead me to believe, in the absence of positive evidence of gonorrhea and with the multiple joints involved, that the condition is probably rheumatoid arthritis, the general resistance being lowered because of recent infection with syphilis.

DR. S. E. SWEITZER, Minneapolis: This young lady has infectious arthritis and more than likely has gonorrhea. The complement fixation test was negative, it is true, but it is not always positive for patients with gonorrhea anyway. Smears were negative also, and this is inconclusive.

Dermatitis Herpetiformis. Presented by DR. S. E. SWEITZER, Minneapolis.

O. D., a white girl aged 9 years, has a generalized, mildly pruritic eruption consisting of blebs and erythematous areas, which developed after an attack of scarlet fever three years ago. The eruption was diagnosed as "pemphigus." Since the onset the eruption has decreased and symptoms of pruritus grown less marked.

A symmetric eruption involves the axillas, wrists, hands and vulva. The lesions are grouped tense vesicles and blebs, surrounded by erythematous zones. The fluid content of the vesicles at the onset is clear, later becoming purulent if the vesicles are not ruptured. Other areas of skin show hyperpigmentation and depigmentation, representing sites of previous lesions. Nikolsky's sign is not present. Histologic sections are shown.

DISCUSSION

DR. LOUIS A. BRUNSTING, Rochester: Dermatitis herpetiformis is rare in childhood, and a number of features in this case are atypical. Urticarial lesions are absent. On the hands there are infected undermined pustules such as are seen in acrodermatitis continua.

DR. H. E. MICHELSON, Minneapolis: I think those of the members who attended the clinic in San Francisco were impressed by the discussion's bringing out the facts that dermatitis herpetiformis can be different in childhood, that the lesions often look like pemphigus and that the prognosis is better. Germanin has been used in such cases with success, but I do not know if I would recommend it unless the condition was not responding to any other medicament.

DR. S. E. SWEITZER, Minneapolis: This patient had a group of lesions on the back. I have had her under observation for some time. At the moment she has lesions in the axilla.

Lichen Urticatus. Presented by DR. S. E. SWEITZER, Minneapolis.

M. K., a boy aged 8 years, has had severe itching of the face, arms, neck and legs since an attack of smallpox at the age of 1 year. He has had exacerbations and remissions since the onset. The itching is worse in the winter. He has used black salve (a coal tar preparation) without relief; saponated solution of cresol has given some relief.

The patient appears to be somewhat malnourished and of small stature. Over the face, neck, arms and legs are many excoriations and pigmented scars. The skin over the arms is slightly lichenified.

DISCUSSION

DR. H. A. CUMMING, Minneapolis (by invitation): The general impression given by Erich Urbach in his recent treatment of his subject (Skin Diseases and Nutrition, Including the Dermatoses of Children, Vienna, Wilhelm Maudrich, 1932) is that in most cases this condition is the result of one or many hypersensitivities —in other words, is on an allergic basis. That was challenged by F. Hamburger, who expressed the opinion that it was usually the result of insect bites.

Urbach, on the basis of 225 closely observed cases, attempted to substantiate his opinion by showing fairly good results from strict allergic management. It is interesting to note that most of the patients are children who seem to be malnourished and come from the lower walks of life.

DR. ELMER M. RUSTEN, Minneapolis: These children have one striking characteristic: extensive cutaneous reactions to scratch and intracutaneous tests. The elimination of the causative substances from the diet or environment does not alter signs or symptoms. Dietary imbalance—not necessarily deficiency—appears to be more important than contact with specific allergens, and when this is corrected the patients improve.

DR. STEPHAN EPSTEIN, Marshfield, Wis. (associate member): The papular eruption involving the arms and legs of a boy of 7 years could be called prurigo mitis of Hebra as well. There are enlarged glands in both axillas. The differentiation may be of minor significance, as both conditions probably belong to the same allergic group.

DR. CARL LAYMON, Minneapolis: The remarks of Drs. Epstein and Rusten agree with the conclusions drawn by Abraham Walzer and Max Grolnick in their study of papular urticaria (*J. Allergy* **5**:240 [March] 1934), namely, that the disease is probably atopic. In their experience, however, specific therapy based on cutaneous tests was of no avail.

DR. S. E. SWEITZER, Minneapolis: In Europe prurigo mitis occurs, but here it is not seen. In our cases the picture fits in with lichen urticatus. This condition looks to me like severe lichen urticatus. It was three times as bad two weeks ago.

DR. H. E. MICHELSON, Minneapolis: I think lichen urticatus should be looked on as a papular urticaria of childhood. That does not explain the causation, it is true, but the clinical appearance is definite. The lesions are conic papules capped by a small vesicle. The patient in scratching excavates the tip of the papule, which releases the fluid in the small vesicle. The secretion coagulates into a tiny crust, so that one usually notices crusted papules. The lesions are on the extensor surface of the forearms and on the face, especially the forehead, as well as in the anterior part of the tibial region. In cases of true prurigo these lesions are present and in addition lichenification, pigmentation, eczematization and adenitis. Prurigo is a much more severe disease and seems to be as closely related to dermatitis herpetiformis as to atopic eczema. Certainly the subjective symptoms are the most striking. Prurigo is chronic and seems to affect the entire being. The patients are often retarded mentally and become a type which, at least in Vienna clinics, used to be recognized as characteristic for prurigo.

A Case for Diagnosis (Light Sensitivity Dermatitis?). Presented by DR. S. E. SWEITZER, Minneapolis.

M. T., a Negress aged 63, first noticed itching of her right arm about six months ago. Gradually various other areas became involved, especially after exposure to sunlight during fishing. The itching became severe over the dorsa of both forearms and around the neck over the exposed parts. She noticed that her skin was becoming darker over the itchy areas. She had received two injections of neoarsphenamine for syphilis previous to the onset of this trouble. Many salves and lotions were used without relief. Her past health had been excellent.

Darkly pigmented rather sharply outlined areas on the flexor and extensor aspects of both arms extend from just above the elbow to the wrist. The skin shows some thickening and accentuation of the lines of cleavage but no other lesions except a few small scars, probably from excoriations. A similar involvement extends around the neck, limited to the exposed surfaces.

Histologic sections are shown.

A Case for Diagnosis (Eczema Due to Light? Lupus Erythematosus?). Presented by DR. H. E. MICHELSON, Minneapolis.

E. B., a white woman aged 39, first noticed an eruption on the face when she was 7 years old. Until about 1930 the lesions disappeared during the winter. Since then they have been practically constant, improving but slightly during the winter. The lesions are pruritic. Only the face, the "v" area of the neck and the arms have been affected. The eruption seems to flare up after exposure to the sun's rays. But little improvement has followed roentgen therapy and application of various light-protective pastes.

The lesions are located on the face, neck and arms. They appear as scaly erythematous patches. There is no actual vesiculation, atrophy, scarring or follicular dilatation.

Studies of the urine for porphyrin by Dr. Cecil Watson gave negative results on two occasions.

DISCUSSION ON PAPERS OF DRS. SWEITZER AND MICHELSON

DR. S. E. SWEITZER, Minneapolis: I thought the first patient might have arsenical eruption. She had had two injections of neoarsphenamine in August and another in October, but she had had the eruption before that time. Localization was around areas of the neck.

DR. ELMER M. RUSTEN, Minneapolis: The Negress has a history of an increase in the eruption on exposure to light, which is uncommon. It is not probable that this factor alone caused the dermatitis; more likely it only irritated a previous eruption, probably a contact dermatitis.

DR. STEPHAN EPSTEIN, Marshfield, Wis. (associate member): As far as the first case is concerned, I recall the case of M. Juon (*Arch. f. Dermat. u. Syph.*

156:355, 1928), of the simultaneous development of hypersensitivity to light, to neoarsphenamine and to mercury after antisyphilitic treatment. I should like to know whether analogous cutaneous tests have been performed in this case. The second case appears to me to be an instance of typical prurigo aestivalis (summer prurigo). Experimental studies in cases of that disease not infrequently have been confined to the hypersensitivity to light alone. It may be worth while to study them with regard to a possible allergic and photoallergic background.

Dr. F. W. Lynch, St. Paul: There is a rather close relation between light sensitivity and a number of metals, including arsenic and mercury. In argyria one frequently observes clinical phenomena only in areas exposed to light, although microscopic studies may demonstrate silver in covered areas without clinical change. In a case recently recorded, a person exposed himself to sunlight frequently during the summer months, with no unusual effect. In the fall he was given injections of pituitary extract for a possible endocrine disturbance. With no further exposure to light, hyperpigmentation developed only on areas not covered by his bathing suit in the summer months, suggesting a clinical relation between the pigmentation and the pituitary medication. In lower animals it has been demonstrated that the middle lobe of the pituitary gland secretes a substance, intermedin, which has much to do with the production of pigment in the skin.

Dr. Louis A. Brunsting, Rochester: The appearance of the eruption suggests papular atopic dermatitis limited to the exposed surfaces, probably conditioned by exposure to light. The various investigative procedures have given no clue to causation. Disturbances of porphyrin metabolism are not concerned in this type of disease. I do not think the condition is lupus erythematosus, in spite of the suggestive histologic picture.

Leukonychia. Presented by Dr. H. E. Michelson, Minneapolis.

R. B., a white youth aged 14, states that his finger nails have gradually turned white during the past six years. He has been nervous since birth and has bitten his nails constantly. The left index finger and right middle finger were first affected.

All the nails of the left hand and the second and third right finger nails are white. The left thumb nail appears to be undergoing a similar change. The toe nails are normal.

Neurologic examination gave negative results.

The patient was adjudged to be of normal or average intelligence by the child psychiatry department.

DISCUSSION

Dr. Carl Laymon, Minneapolis: Leukonychia is usually of the punctate or striate variety, whereas the total type present tonight is extremely rare. The whiteness is thought by some authorities to be due to the presence of small amounts of air between the nail cells, while others believe it due merely to abnormal cornification. The etiologic factors of leukonychia totalis remain unknown.

Leukemid. Presented by Dr. S. E. Sweitzer, Minneapolis.

M. L., a white woman aged 64, first noticed an eruption on her legs in August 1938. The itching was severe, and the patient was extremely uncomfortable. She was treated with local applications, and the eruption cleared completely. About six weeks ago it again appeared on her legs. It has been present since that time.

Two years ago the patient was found to be suffering from chronic lymphatic leukemia, for which she was given roentgen therapy. Otherwise her past health has been fair.

There are many scattered macular, papular and excoriated lesions over both legs and thighs. The older lesions are light brown and have a dry branny scale.

The new lesions are papulovesicular with an erythematous base and are extremely itchy. The lesions vary in size from that of the head of a match to that of a nickel.

Histologic sections are shown.

The urine was normal. The white blood cell count was 92,800, with 20 per cent polymorphonuclears, 75 per cent lymphocytes, 3 per cent monocytes and 2 per cent eosinophils. Study of sections established a diagnosis of chronic lymphatic leukemia.

DISCUSSION

DR. PAUL A. O'LEARY, Rochester: I disapprove of calling the lesions leukemid, because to me this picture is one of toxic pruritus such as is encountered in patients with leukemia. When the histologic picture of an excised cutaneous lesion does not show the characteristic cytologic changes of leukemia, the use of the term leukemid is unwarranted, and as I understand it, the histologic picture in this case was not specific.

DR. F. W. LYNCH, St. Paul: The term leukemid was introduced by Audry forty years ago, when he applied it as a descriptive term for those eruptions associated with leukemia without the clinical characteristics of true leukaemia cutis and a specific histologic structure. Since that time the terms leukemid and leukaemia cutis have been used, respectively, to describe the nonspecific and the specific eruption. In recent years there has been a tendency to disregard this sharp division between the eruptions, since in a few cases of a nonspecific microscopic structure the clinical appearance has been characteristic of leukaemia cutis, and in other cases of a clinically nonspecific picture the microscopic structure has been that of true leukaemia cutis. The two terms should probably be retained for convenience in discussions, but it seems likely that the dividing line is not a sharp one.

Reticuloendotheliosis. Presented by DR. S. E. SWEITZER, Minneapolis.

H. K., a white boy aged 3 years, first had some difficulty in walking about six months ago. His toes would point in, and he would stumble and fall. An eruption developed over his body about the same time. According to the parents, it began as small blisters, which ruptured when scratched. The eruption became generalized soon after the onset and has remained so. The child has never been a good eater, which fact has been especially noticeable during the past two or three months. He has lost considerable weight during his six months of illness, and difficulty in speaking has developed. The family history is irrelevant.

The patient is poorly nourished, pale and unresponsive. Many yellowish brown firm papular lesions are scattered over his body. They are especially numerous on the face. Many of the lesions have been excoriated, and some of the more recent ones have a hemorrhagic appearance. They vary in size from that of the head of a pin to that of a peanut.

The urine was normal. The hemoglobin concentration was 52 per cent, the white blood cell count 15,250 and the red blood cell count 3,440,000; the differential count showed 81 per cent polymorphonuclear cells, 16.5 per cent lymphocytes, 0.5 per cent eosinophils and 2 per cent monocytes. The blood sugar value was 70 mg., calcium 11.5 mg. and the cholesterol 172 mg. per hundred cubic centimeters. The spinal fluid showed protein 45 mg., sugar 48 mg. and no cells. The Wassermann reaction of the blood was negative. Cultures of material from the nose and throat were negative. The Mantoux test with a 1:10,000 dilution was negative. Roentgen studies of the chest and lungs gave negative results. The bones showed: (1) erosion or cystic appearance in the sixth rib on the right side, consistent with a tumor; (2) a destructive appearance on the right side of the pelvis, with destruction of the acetabulum and no new bone formation; (3) destruction of the distal portion of the shaft of the left femur, rarefaction with periosteal new bone formation and pathologic fracture of the left femur; (4) destruction of the entire first lumbar vertebra, and (5) normal hands, wrists, arms and skull.

The histologic picture of the skin seemed to be consistent with a diagnosis of reticuloendotheliosis.

DISCUSSION

DR. F. W. LYNCH, St. Paul: I think this case deserves discussion because in few such cases is the diagnosis made ante mortem. The studies in this case are complete, and the diagnosis appears to be correct. In these microscopic sections, as in the cases I observed at the University of Minnesota Medical School, one cannot differentiate with certainty between monocytic leukaemia cutis and reticuloendotheliosis, but the hematologic studies ruled out the former disease.

A Case for Diagnosis (Possible Besnier-Boeck-Schaumann Disease?). Presented by DR. H. E. MICHELSON, Minneapolis.

R. A., a white woman aged 37, noticed gradual formation of nodules over her body after treatment for a cutaneous disease diagnosed as scabies about one year ago. Some of these lesions have since disappeared. She has suffered from malaise, anorexia and fatigue since that time and has lost about 30 pounds (13.6 Kg.). There has been no pain, itching or burning. She was treated with a sulfur ointment at first and later with zinc oxide ointment.

The patient appears greatly emaciated. Her posture is poor, and she walks with a limp. There is a generalized increase in cutaneous pigmentation, especially on the exposed surfaces. On the face, trunk and upper extremities there are numerous nodular and plaquelike lesions, which are reddish brown and appear to be located over those portions of the body which are exposed to pressure. There are no lesions on the lower extremities. Pathologic sections were shown.

Urinalysis showed no abnormality. The Wassermann reaction of the blood was negative. The calcium, sugar and phosphorus contents of the blood were normal. The cholesterol content was 250 mg. per hundred cubic centimeters. The basal metabolic rate was +73 per cent. Gastric analysis showed free hydrochloric acid following administration of histamine. Roentgen studies of the chest were normal. The hands showed multiple sharply circumscribed punched-out areas. The long bones were normal.

DISCUSSION

DR. J. F. WILSON, Minneapolis (by invitation): It has never been possible to make a positive diagnosis in this case. The original biopsy specimen, from the shoulder, did not present a typical histologic picture of sarcoid. The second biopsy specimen, taken from the chin, showed a picture considered by Dr. E. T. Bell, of the pathology department, to be consistent with the diagnosis of sarcoid. An interesting feature in this case is the basal metabolic rate. In July, when first studied, it was +56 per cent. A determination on October 21 showed it to have increased to +73 per cent. There were no evidences of thyroid disturbance.

DR. PAUL A. O'LEARY, Rochester: I agree that this patient does not have Schaumann's disease. I have seen grapelike masses such as she presents in a patient who had cutaneous metastases from hypernephroma.

DR. CARL LAYMON, Minneapolis: At the first examination diagnoses of sarcoid, metastatic malignant growth, lymphoblastoma and some form of lipoidosis were considered. It was impossible to aline the process with ordinary xanthoma, on account of the lack of foam cells and intracellular lipoid masses in any of the sections. The nodules on the hands and the histologic picture are not unlike those in the case of extracellular cholesterosis observed at some of the meetings of this society several years ago (ARCH. DERMAT. & SYPH. **35**:269 [Feb.] 1937).

DR. H. E. MICHELSON, Minneapolis: I am not willing to commit myself on the diagnosis in this case. However, I am inclined to think that the diagnosis of sarcoid is incorrect. The sections certainly are not those of sarcoid. There is some resemblance to a form of fat disturbance.

A Case for Diagnosis (Pigmentation Due to Copper?). Presented by DR. S. E. SWEITZER, Minneapolis.

J. Z., a white man aged 59, first noticed a slight discoloration of his skin about one and one-half years ago. He noticed this after he had had a hernia treated

by the injection method. As time went on his skin became darker. There was no itching or discomfort of any kind. He took a moderate amount of "Harlem oil" for some urinary complaint. In July 1938 he suffered a pathologic fracture of the tibia and was admitted to the hospital because of its nonunion. His past health had been good, and he had worked at his occupation as a horse handler for years.

There is a rather diffuse mottled appearance of the skin, more pronounced on the face, neck, hands and arms. The discolored areas are bluish gray and discrete and vary in size from that of a pinhead to that of the head of a match. There are no excoriations or other cutaneous blemishes.

Both excision and punch biopsy were done. The histologic picture was that of degenerated oval hyaline masses, well circumscribed in the upper part of the cutis.

Spectroscopic examination of the skin showed a copper line of 0.1 to 1.8 per cent (within normal limits).

Urinalysis showed no abnormality. Bence Jones proteins, alkaptone bodies and melanin were absent. A blood count showed 74 per cent hemoglobin, 3,930,000 red cells and 9,050 white cells, with a differential count of 61 per cent neutrophils, 31 per cent lymphocytes, 6 per cent monocytes and 2 per cent eosinophils. The Wassermann reaction was negative. The blood calcium content was 10.8 mg., the phosphorus 3.8 mg. and the phospholipin 4.9 to 4.1 units. Roentgenograms revealed cystic changes of both femurs, but showed normal skull, humeri, ribs and lungs.

DISCUSSION

DR. PAUL A. O'LEARY, Rochester: The appearance of such unusual pigmentation in a person who does not work with copper or other metals brings to mind patients with lymphosarcoma who have manifested similar pigmentation about the face. The fact that he has had the pigmentation for a year and a half and does not as yet have palpable adenopathy does not as yet eliminate lymphosarcoma as a possible cause of the discoloration.

DR. LOUIS A. BRUNSTING, Rochester: This is pigmentation of a most unusual type. Perhaps ochronosis should be given more consideration.

DR. CARL LAYMON, Minneapolis: The patient worked with tar for several years. Could this dermatosis fit in with so-called lichenoid melanodermatitis due to contact with tar?

A Case for Diagnosis (Pustular Psoriasis? Seborrheic Dermatitis? Avitaminosis?). Presented by DR. S. E. SWEITZER and DR. ELMER M. RUSTEN, Minneapolis.

M. S., a white woman aged 28, has had trouble with her skin since the age of 6 months. The eruption was always more severe during the hot summer months, although she was unable to state any definite time at which it became aggravated. For six years she has suffered from seasonal asthma, which has been associated with hay fever during the last two years. The onset had always been during the last week of July and the termination with the coming of frost.

There were no allergic diseases in members of the family except the maternal grandfather, who had asthma. During treatment for her hay fever this year her cutaneous condition was definitely aggravated by one injection of a solution containing ragweed and sagebrush pollen, although subsequent injections produced no noticeable exacerbation. The patient was hospitalized about the middle of July, after which improvement was rapid under local therapy with ointment of crude coal tar and ultraviolet rays. At the onset of ragweed pollenization (August 15), mild symptoms of hay fever developed, as well as weeping erythematous lesions about the nose, axillas and groin.

There is a generalized thinning of the hair on the scalp, with patches of total alopecia. The skin in these areas is atrophic and smooth. The greater part of her eyebrows is gone, and there is complete absence of eyelashes. Both corneas are cloudy, and there is some ectropion of the left lower eyelid. There are

scattered erythematous scaly lesions over the face. The axillas, popliteal spaces, feet and groin show a rather diffuse weeping erythematous eruption, which is scaly and crusting in spots. Fissures in the natural folds of the skin are painful. Several of the finger nails show a watch crystal deformity.

Histologic sections are shown.

DISCUSSION

DR. LOUIS A. BRUNSTING, Rochester: I think this condition is seborrheic eczema in the broad sense, and there is superimposed pyogenic involvement.

DR. F. T. BECKER, Duluth (by invitation): Recent German work has brought out a new vitamin, H, which appears to be of therapeutic value in seborrhea. This was first introduced as the X factor and was apparently specific for rat seborrhea. E. Györgyi (in von Pfaundler, M., and Schlossmann, A.: Handbuch der Kinderheilkunde, vol. 10, p. 45, Leipzig, F. C. W. Vogel, 1935) has found this substance efficacious in the treatment of seborrheic eczema of infants and adults. Although vitamin H has not been isolated in pure form, it is believed to be found in large quantities in beef liver, milk, potatoes, brewers' yeast and bananas. Vitamin H is necessary for the proper maintenance of normal cutaneous fat metabolism. A reduction in the intake of fat and egg yolk and the addition of foods containing vitamin H was efficacious in Györgyi's hands.

DR. ELMER M. RUSTEN, Minneapolis: I agree with Dr. Brunsting. This patient has neurodermatitis in seborrheic areas, and there is a definite relation of her symptoms to her hay fever and menstrual disturbances. When either are severe the cutaneous symptoms increase.

Panniculitis. Presented by DR. S. E. SWEITZER, Minneapolis.

C. P., a woman aged 70, first noticed several discolored areas on the dorsa of her hands one month ago. These areas were not tender and did not itch. Two weeks ago red swollen hot moderately painful areas began to develop in the skin below the right knee, and soon afterward similar areas appeared around both elbows. These have slowly enlarged and new ones appeared over the extensor surfaces of the tibias and one over the right buttock. There has been a sustained temperature varying from 99.6 to 102 F. There was no history of ingestion of drugs.

There are swollen hot tender purplish red areas on the extensor surfaces of both forearms and some superficial violaceous areas on the dorsa of the hands and the anterior surface of the legs.

Histologic sections are shown.

DISCUSSION

DR. PAUL A. O'LEARY, Rochester: The diagnosis of chronic nonsuppurative panniculitis seems warranted. The process is apparently of a mild type, although the lesions are numerous.

LOS ANGELES DERMATOLOGICAL SOCIETY

KENDAL FROST, M.D., *Chairman*

Dec. 13, 1938

CHARLES R. CASKEY, M.D., *Secretary*

A Case for Diagnosis (Iododerma Due to Iodized Salt?). Presented by DR. H. C. L. LINDSAY.

R. H. M., a white man aged 65, a pharmacist, has an eruption located principally on the legs, which began about two months ago. It consists of multiple shallow ulcers covered with brownish crusts. Each lesion is surrounded by a purplish

red inflammatory areola; some of them are discrete papules with crateriform centers. Old lesions which have healed have left scars. There is no history of ingestion of drugs other than the use of iodized salt. A physical examination showed no abnormality except numerous decayed teeth. A blood count showed 59 per cent hemoglobin and 4,000,000 red cells. The white blood cell count was normal. A urinalysis showed albumin and occult blood. The stool also revealed occult blood. The Wassermann reaction was negative.

DISCUSSION

DR. L. F. X. WILHELM: I think the whole picture might be explained on the basis of iododerma. I saw little resemblance to molluscum contagiosum or tuberculid.

Fig. 1.—Iododerma from iodized salt.

DR. SAMUEL AYRES JR.: I think that a diagnosis of molluscum contagiosum should be considered. However, I think that tuberculid is more likely. I have seen atypical molluscum with considerable inflammatory reaction. I understood the patient to say that he picks out the lesions and they heal. Of course, a histologic examination would solve the problem.

DR. H. S. CAMPBELL: I subscribe to the diagnosis of iododerma. I do not think the picture looks anything like molluscum contagiosum and do not believe it could justifiably be confused with it, even when secondarily infected

DR. WILLIAM H. GOECKERMAN: Clinically, I think it could be iododerma. I cannot say much for tuberculid. There is a decided inflammatory halo around each lesion. I believe molluscum contagiosum should be considered. I made a

brief search for lesions of molluscum but could find none. Cases of iododerma should be studied in detail, such measures as examination of the urine for iodides being included.

DR. H. C. L. LINDSAY: From the point of view of Dr. Ayres, one of the lesions looks much like molluscum contagiosum. It is a pearly papule with a definite center, but the other lesions look more like bromide granulomas. According to the history the man has taken no bromides as medicine, but he has been taking iodized salt continuously with his meals for several years. The lesions are inflammatory, and the general appearance coincides with that of a halogen rash, but in this instance with more of a bromide than an iodide configuration.

NOTE.—Chemical examination of both the urine and the feces for iodine gave negative results. The histologic observations were inconsistent with the picture of molluscum contagiosum.

Granuloma Coccidioidale (Four Cases). Presented by DR. HARRY P. JACOBSON.

CASE 1.—S. L., a white American man aged 38, is a mechanic engaged in airplane construction. On April 2, 1934, he struck his left shin on a piece of steel. A hematoma formed at the site of the injury. On April 9 he wrenched his left shoulder, and three days later a swelling appeared over the left sternoclavicular joint. Incision over this swelling revealed no pus. He continued work until April 27. On May 22 he was admitted to the dermatologic service of the Los Angeles County Hospital. At this time there were abscesses at the site of each of the aforementioned injuries. From each a small amount of pus was draining through a small sinus. This pus contained typical double-countoured endosporulating organisms. Roentgenograms showed considerable destruction of both clavicles and the left shoulder and pleuropulmonary coccidioidal granuloma. Coccidioides immitis was obtained from both abscesses.

His treatment at that time was with large doses of iodides, colloidal copper intramuscularly, coccidioidin subcutaneously and cod liver oil. The fistulas were dressed daily with cupric sulfate solution. In November 1934 he was discharged to the outpatient clinic. In April 1935 an autogenous vaccine was prepared. This was given intravenously and all other medication discontinued. Under this plan progress was rapid. This patient has had no treatment since Feb. 8, 1938, and the disease seems completely arrested.

CASE 2.—E. G., a white man aged 19, was admitted to the Los Angeles County Hospital in the fall of 1936 on account of a peritonsillar abscess from which drained considerable pus. The abscess was traceable to lymph nodes in the anterior triangle of the neck, which in turn drained through a cutaneous lesion in that region. Improvement was so rapid under treatment with coccidioides vaccine that he left the hospital and disappeared for three months. In the middle of March 1937 he returned, with extensive involvement of the left sternoclavicular region and all the soft tissues and overlying skin. From the abscess typical C. immitis was recovered on culture. He has been treated with coccidioides vaccine and cod liver oil. He has had no treatment since May 11, 1938, and the condition appears clinically to be completely arrested.

CASE 3.—A. H., a white American man aged 44, a miner, gave a history of an injury to the back followed by an abscess in the bruised area two weeks afterward. C. immitis was found in the pus from the incised abscess. He was in the Los Angeles County Hospital from June 2 to Aug. 15, 1936, after which he disappeared from observation. Treatment consisted of rest in bed, administration of cod liver oil and colloidal copper and intramuscular and subcutaneous injections of coccidioides vaccine. He also had treatment for cerebrospinal syphilis. In

September 1936 he was readmitted to the hospital, with extensive involvement of the right inguinal region. Roentgenograms showed no involvement of the lumbar vertebrae. For the past three months he has been given antisyphilitic treatment. The only treatment he has received for the coccidioidal condition has been intravenous injections of coccidioides vaccine. Those who saw this patient at the meeting of this society on April 12, 1938, can appreciate the improvement. Figure 3 shows a healing process.

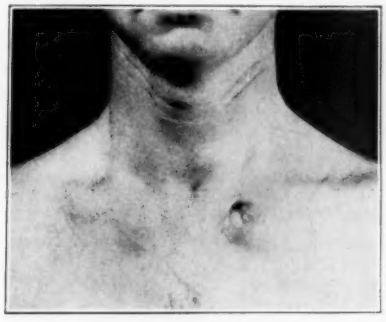

Fig. 2.—Clavicular coccidioidal involvement, with soft tissue ulceration and healing.

Fig. 3.—Inguinal coccidioidal ulceration in the process of healing.

CASE 4.—J. O., a white American man aged 61, a barber, gave a history of pain in the right hip for two years. In January 1937 he was admitted to the hospital with a retropharyngeal abscess, from which C. immitis was recovered. He was given several transfusions immediately after admission to the hospital for severe secondary anemia. Roentgenograms of the chest revealed an old bilateral apical involvement suggestive of tuberculosis. Roentgenograms of the right extremity and vertebrae revealed rarefaction of the fourth and fifth cervical vertebrae and apparent rarefaction of the sacrum at its right ala and the sacroiliac

joint. Comparative study in October 1937 showed no active pathologic changes. Treatment consisted of injections of coccidioides vaccine, subcutaneously at first, then intravenously; complete rest, and administration of halibut liver oil. He has received no treatment since June 9, 1938, and has since returned to full time work.

DISCUSSION

DR. H. S. CAMPBELL: These reactions are so spectacular that they savor of an advance in the therapy of a decidedly resistant and in large measure hopeless condition, in which therapeutic claims often indulged in are invariably found wanting. I should like to know whether these cases are the cream of the group or whether they are representative of what is occurring in the whole group. The disease definitely tends to fluctuate in the deeper systemic type, which these cases represent. As I say, if this represents what is occurring in them all, in direct response to this intravenous therapy, then I think Dr. Jacobson has struck an advance note in the therapy of this disease. What reaction is obtained from the vaccine?

DR. H. C. L. LINDSAY: I believe that Dr. Jacobson should be highly commended for his tenacious efforts in curing some of these patients. I should like to ask how the appearance of a scar of coccidioidal granuloma would differ from a scar of tuberculosis cutis or syphilis.

DR. L. F. X. WILHELM: Along the lines of Dr. Campbell's first statement, I should like to suggest that Dr. Jacobson present these patients at six month intervals so all the members can satisfy themselves as to the permanence of the cure obtained.

DR. HARRY P. JACOBSON: These 4 cases of coccidioidal granuloma were picked at random to illustrate the results of treatment of the disease with vaccine intravenously. To date I have a series of 20 odd cases in which this method has been used, with results similar to those shown in these 4. In the past three years or thereabouts I have lost only 1 patient with coccidioidal granuloma, one with extensive meningeal involvement. I have experienced no difficulty with my ambulatory patients, who receive the injections of vaccine in the office; they are instructed to go home immediately and lie down for the duration of the chill. The focal reactions are fairly severe, affecting alike the systemic and the cutaneous lesions.

I am greatly impressed by the results of this form of therapy. The method is applicable not only to coccidioidal infections but to some of the other systemic mycoses. In 1 case of actinomycosis with extensive involvement of the liver and lungs, in which there had been no response to treatment with iodine, thymol or roentgen rays, the response to intravenous vaccine therapy was most satisfactory. The vaccine should preferably be autogenous, but when that is not possible, a polyvalent vaccine including a great many strains is advantageous.

Jan. 10, 1939

Carcinoma en Cuirasse. Presented by DR. SAUL S. ROBINSON.

J. M., a white woman aged 67, had a radical mastectomy in 1935 for adenocarcinoma. In 1935 and 1936 she had twenty-six roentgen treatments over the area. In March 1938 the left breast was removed for metastatic carcinoma. Soon after this nodules and thickened red skin appeared over the right side of the thorax and right arm. This involvement of the skin has progressed, until now there are induration, nodules and ulcerations on the right side of the thorax and on the right arm and forearm. The patient was presented before this society on

Nov. 9, 1937, by Dr. C. R. Halloran (ARCH. DERMAT. & SYPH. **37**:1061 [June] 1938) and is presented at this time to show further extension of the disease.

DISCUSSION

DR. SAMUEL AYRES JR.: This condition looks like that in a case I presented about one year ago and, I think, belongs in the group of carcinoma erysipeloides following removal of mammary cancer, with recurrence in the other breast and apparent lymphatic spreading of cancer cells. This acute process coming on in the past few months is an inflammatory process in the lymph spaces. The prognosis is altogether bad.

DR. WILLIAM H. GOECKERMAN: I should like to emphasize that in this type of carcinoma the erysipeloid character of the lesion is the result of infiltration of the lymphatic vessels by carcinoma cells rather than actual infection.

DR. SAUL S. ROBINSON: The principal reason for presenting this patient was the extensive involvement of the right arm and forearm. In most of the cases reported the lesion has been on the thorax. This patient's condition has been progressing rapidly, with dissemination of the carcinomatous tissues.

A Case for Diagnosis (Herpes?). Presented by DR. ANKER K. JENSEN (by invitation).

R. F., a white American laborer aged 32, has a lesion on the side of the neck, which followed a cut by a razor three years ago. There has been a slight amount of spreading since that time. At no time has the lesion completely healed. There has been no similar lesion elsewhere on the body. There are few subjective symptoms. The lesion on the right side of his neck is about the size of a dollar. The skin over this area is atrophic and presents a shiny appearance. Under this thin atrophic layer there is a small amount of serous exudate. The borders are well outlined. The man is in good health and has an excellent blood count. Examinations of the urine and the blood showed no abnormalities. The treatment has consisted of six ¼ skin unit doses of roentgen rays and applications of different ointments and paints. He has had four injections of smallpox vaccine and eight injections of snake venom. No improvement was noted from any of these.

DISCUSSION

DR. NELSON PAUL ANDERSON: Dr. Ayres and I have under observation a group of cases to which I believe Dr. Jensen's case belongs. We have observed some 4 or 5 cases of a herpetiform eruption, which this condition undoubtedly is, appearing especially about the neck. In some there are lesions about the groins. The condition is extremely resistant to therapy and does not seem to respond to roentgen irradiation. It spreads by peripheral extension a great deal as impetigo does, with undermining of the edges. This lesion is larger than any single one that we have seen. We have more or less tentatively called the condition "recurrent herpetiform dermatitis repens." In several cases the possibility of a drug as an etiologic factor has been suggested. When this possibility has been traced there has been nothing to it. The histologic slide was not characteristic of any dermatosis, and yet I believe the condition to be a definite clinical entity. I am wondering if any of the other members have seen anything like it.

DR. H. S. CAMPBELL: In view of the persistence, why does Dr. Anderson use the term recurrent herpetiform eruption? The differentiation between the entity in question and simple herpes, one would say, lies mainly in the length of time over which the lesion persists.

DR. WILLIAM H. GOECKERMAN: If Drs. Anderson and Ayres have discovered a new entity I am glad to know of it. I have never seen anything that reminds me of this condition, and I feel that it is a dermatitis artefacta, largely because I cannot place it in any category with definite characteristics. In herpes sometimes the bullae become large as the result of confluence of the vesicles.

DR. SAMUEL AYRES JR.: When I first saw this patient that diagnosis came to my mind. I think it belongs in the group that Dr. Anderson has described. This case is the fifth or sixth that he and I have observed. Characteristic of these cases, as Dr. Anderson has mentioned, is the fact that most of the lesions are about the neck, with a few in the groin. The lesions are herpetiform but not typical of herpes simplex. They present herpetiform vesicles but spread like dermatitis repens with moist borders. They are rebellious to treatment. Some of the patients respond to roentgen therapy and remain well for months, with recurrence in the same place or some neighboring area. We have 2 cases at present, those of a mother aged 70 and a daughter aged 45. I have been puzzled as to how to classify the entity, and the name we thought of suggests the herpetiform appearance combined with the spreading habit of dermatitis repens. I should be much interested in knowing if any one else has seen a condition like this.

DR. H. S. CAMPBELL: My feeling is that this lesion is an artefact, because within the affected part over its central area there are three small areas of definite scar tissue. On the other hand, some time ago I had a patient with a condition similar in certain respects to the one which is being described tonight as a new entity. It was on the right side of the gluteal area; when presented, the lesion was three days old. Although herpetiform, the central zone was devoid of vesicles, being simply reddened. It extended marginally by a narrow band of undermined skin and was resistant to therapy. I remember being somewhat puzzled by it, and I concluded it might be somewhat resistant and atypical recurrent herpes.

DR. S. W. BECKER, Chicago (by invitation): I have never seen an eruption just like this one. Of three areas of activity, at least one showed typical herpetiform vesicles. The plaque contained no hair, and the skin was atrophic, which would be difficult to explain on the basis of herpes simplex. I could get no history of injury, which would suggest neuritic dermatitis, as described by me and later by Dr. Ruth Herrick, from the clinic with which I am associated. The patient did state that he had a painful sensation on the inside of the neck on that side, so that the lesion may be neuritic. The alopecia and atrophy may be the result of treatment. I should suggest inoculation of a rabbit's cornea with serum from the vesicles, a procedure which in practically all cases of herpex simplex is followed by encephalitis.

DR. PAUL D. FOSTER: Herpes simplex recurrens has varying periods of latency. It seems possible that the attacks could occur in such close proximity as to appear as one, thereby confusing the picture. The scarring and alopecia present strongly suggest herpes to me. I should suggest vaccination with smallpox vaccine, according to the technic of Abshier and me (ARCH. DERMAT. & SYPH. **36**:294 [Aug.] 1937).

DR. NELSON PAUL ANDERSON: The condition has always looked as it does tonight. What Dr. Goeckerman brought out was what I have thought a number of times. The man has not worked for three years because of this lesion on his neck. I told him he was able to go to work, but he was not pleased. The next time I saw him it was at the clinic of White Memorial Hospital. When I see him again I shall put on an occlusion dressing and see if it makes any change.

Pityriasis Rosea (of Eight Months' Duration). Presented by DR. NELSON PAUL ANDERSON.

E. R., a white American woman aged 23, has an eruption which began eight months ago as a pink scaly area on the left breast. About two months ago a similar area appeared on the left hip. In the past six weeks about a dozen new lesions have appeared on the trunk. At this time the "mother spot" appears as a relatively large plaque with an erythematous annular oozing eruption larger than a dollar on the left breast.

DISCUSSION

DR. L. F. X. WILHELM: I think the condition is pityriasis rosea. It is unusual for the herald patch to be present six or eight months before the rest of the eruption appears.

DR. WILLIAM H. GOECKERMAN: I cannot agree with the diagnosis of pityriasis rosea without further study. There is the possibility of beginning mycosis fungoides.

DR. SAMUEL AYRES JR.: I think that the extremely long duration of the initial patch and the present rather moist appearance would make one consider mycosis fungoides. The whole picture is certainly unusual for pityriasis rosea.

DR. M. NORRIS ROSENBERG: With the oozing it may be beginning Paget's disease.

DR. CHARLES R. CASKEY: I cannot agree with the diagnosis as presented. My interpretation is that the lesion on the breast is of fungous origin (possibly monilial) and that the other lesions are ids.

DR. H. S. CAMPBELL: I cannot accept this case as one of pityriasis rosea, either clinically or historically, as now, after ten weeks, new lesions of doubtful character are still appearing. The oozing and inflammation over the areola of the breast, where the early lesion was situated, are due to local medication. My impression is that the condition may be either parapsoriasis or commencing mycosis.

DR. NELSON PAUL ANDERSON: The main point in presenting the patient was to show that this disease, among all the acute inflammatory dermatoses, presents the most varied picture. There are hemorrhagic types and papular and macular types. I think that with the large macular type of pityriasis rosea one sees the most variations and the lesions persist the longest. The textbooks state that the duration of the disease is six or eight weeks if no treatment is given. I think some of these large macular types will last six or eight months or even a year if no treatment is given, and sometimes their persistency in spite of treatment is astounding.

Neurofibroma (?) Associated with Dermatosis Papulosa Nigra. Presented by DR. MAX J. WOLFF.

C. G., a Negro aged 39, has lesions which have appeared gradually for about twelve years. A few have "come to a head," and the patient was able to express a "core." At this time he presents numerous discrete soft dome-shaped nodules varying in size from that of a pea to that of a cherry, some of which invaginate on pressure. Most of them are of the same color as the surrounding skin, and a few are darker. Most of the lesions are about the lower part of the trunk, but a few are present about the infrascapular region. There is one large movable almond-shaped subcutaneous nodule on the extensor surface of the left arm, above the elbow. Scattered over the face are flat-topped black papules varying in size from that of a pinhead to that of a match head.

DISCUSSION

DR. M. NORRIS ROSENBERG: I got the impression from the lesions and the associated pigmentation, with the associated low intelligence quotient, that the patient has Recklinghausen's disease.

DR. S. W. BECKER, Chicago (by invitation): My first impression was that the eruption was multiple steatocystoma, described by O. S. Ormsby and C. W. Finnerud in the ARCHIVES (**22**:822 [Nov.] 1930). The lesions are in the seborrheic areas, a distribution I have never seen for Recklinghausen's multiple neurofibromatosis. The section showed only normal connective tissue, not the glia-like tissue of neurofibroma. I should suggest that a histologic specimen be bisected, so that the center of the tumor will be sectioned.

DR. MAX J. WOLFF: There is a section under the microscope. It is not suggestive of neurofibroma.

Book Reviews

Syphilis and Yaws in Guatemala: Chapter 10 of a Medical Survey of the Republic of Guatemala. By George Cheever Shattuck, Assistant Professor of Tropical Medicine, Harvard School of Public Health, with the collaboration of J. C. Bequaert, M. M. Hilferty, J. H. Sandground and S. D. Clark. Price, $2.50. Publication 499, Carnegie Institution of Washington, 1938.

The chapter under consideration deals with the incidence of syphilis and yaws in the natives of Guatemala. The population consists mainly of two races—the pure-blooded and nearly pure-blooded descendants of the aboriginal Indian tribes and the Spanish-Indian crossbred or mixed races, called Latins. Based on clinical and serologic investigations, the author (in collaboration with Dr. Helen Curth) found that the incidence of syphilis is nearly twice among the Latins what it is among the Indians. The proportion of complete clinical latency is high in both races but higher in the Indians. In the latter there is conspicuous scarcity of clinical evidence of syphilis, pointing toward probable immunologic peculiarities inherent in aborigines.

In Guatemala City the survey indicated a high percentage of syphilis. Neurologic lesions are not rare. Among the native Indian races a survey made by Dr. Estevez showed only 1 per cent of infections. In the highlands of Guatemala the incidence of syphilis fluctuates between 2.7 and 10 per cent among the Latins and the Maya Indians. The disease runs a mild course, indicating that the natives possess an unusual degree of inherent racial or acquired and inherited resistance toward the infection.

Yaws is so rare in Guatemala that newly discovered cases are regarded almost as clinical curiosities. Incidentally, Dr. Shattuck has had a broad experience with yaws in the Philippine Islands, Brazil and Africa, so that the much discussed problem of differential diagnosis did not play a role in the compilation of the data here presented.

The author comments on the incidence and clinical course of syphilis in various countries and among different races of the world. Over a dozen well constructed and easily read tables form a supplement to the chapter.

The monograph has a cardboard binding and is printed in large type on heavy paper. In addition to chapter 10, nearly all the contents of the monograph are of great interest to dermatologists and syphilologists.

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